IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF
MISSISSIPPI, WESTERN DIVSION

FRED BECK, ET AL.,	)	
	)	
Plaintiffs,	)	No. 3:03C0V60-P-D
	)	
vs.	)	
	)	
KOPPERS, INC., ET AL.,	)	
	)	
Defendants.	)	
_____	)	

JAMES DAHLGREN, M.D.
Santa Monica, California
Tuesday, May 10, 2005
Volume IV

Reported by:
DIANA JANNIERE
CSR NO. 10034
L.A. JOB No. 910792

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KOPPERS, INC., ET AL.,)
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Defendants.)
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DEPOSITION of JAMES DAHLGREN, M.D., Volume IV, taken on behalf of Defendants at 1700 Ocean Avenue, Santa Monica, California, beginning at 9:00 a.m., and ending at 5:00 p.m., Tuesday, May 10, 2005, before Diana Janniere, Certified Shorthand Reporter No. 10034.

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EXHIBITS

EXHIBITS (Continued):

Santa Monica, California, Tuesday, May 10, 2005

9:00 A.M. - 5:00 P.M.

JAMES DAHLGREN, M.D.,

having been duly sworn, testified as follows:

FURTHER EXAMINATION

BY MR. HOPP:

Q Back on the record. Doctor, you remember that you are under oath?

A Yes.

Q Dr. Dahlgren, what is your hourly rate in your work with creosote?

A 465 an hour.

Q Do you bill for time devoted by your staff?

A Yes.

Q What is your staff rates? What is a range or average rate for your staff?

A It varies depending on what it is that they are doing.

Q Can you give me a range?

A No, I don't have that in my memory.

Q How much time did you do in your work for creosote cases from inception until now?

A I don't know.

Q Can you give me a range or an estimate?
A I have been working on this case for several years. I don't have that in my memory.
Q Do you know how much you or entities billed into the creosote case so far?
A I don't know.
Q How much of these entities owned by you have been paid on these creosote cases?
A I don't know.
Q Now, as we discussed previously, you mentioned dioxin levels of the blood of residents of Carver Circle?
A Yes.
Q It is an ongoing --
A Yes. That is based on PAH analysis which, I think, I indicated.
Q It depends on the soils and house dust indicated that was collected by others?
A Yes.
Q Do you have reason to believe that the blood level of dioxin in the people from the Carver City neighborhood were higher in the past than they are now?
A I don't know. I mean, you are talking about higher for dioxin levels?
Q Yes.

A Well, there are various reasons why they may have been higher at certain times, at least on an acute basis when they were burning the treated wood.

During that time, they probably would have been higher because of the dioxin being generated by that activity, more than presumably today, they are not treating any treated wood as an energy source. So that would make a difference.

So based on that, one would expect the dioxins to have been higher in years past.

Q Would that have had been an acute basis or during the years which they were breathing treated wood?

A Chronic during the years when they were burning treated wood.

Q Do you know how much higher the dioxin blood levels would have been during the years which they were burning treated wood?

A No, I don't think we have any way of making an estimate about that. All we can say is that it would have been higher.

Q Would it be safe to assume then that, that the dioxin blood levels in the people in the Carver Circle neighborhood would have begun to decrease when the plant stopped burning treated wood?

A Yes. I think they would have experienced a

decrease in their concentrations over time. I mean, the half-life of the dioxins is very long in the body. So it wouldn't have been a very rapid change.

But if the dose is significantly higher and you reduce that dose, it will be reflected over time in the blood level.

Q Do you have any way of estimating or using literature to provide a reference for, by what rate the level of dioxin in the blood in Carver Circle would have decreased over time after the plant stopped burning treated wood?

A Well, the half-life of the various dioxins is variable. The lower the chlorination, the shorter the half-life. The more chlorine atoms attach, the longer the half-life in general.

TCDD, which is the most toxic one, as we discussed before, has a half-life of about seven years based on the studies that have been published.

There is a range. I mean, some people are a little slower. Some people are a little faster, but on average, it is seven years half-life.

That means if your exposure stops completely or is reduced to background, let's say, you will experience a 50 percent decline over a period of seven years in TCDD.

OCDD will probably be longer. It's half-life is probably 12 years. So it would take longer for that one to come down.

And the hepta and the hexadioxins also have a very long half-life. Probably longer than TCDD, but less than OCDD.

Q Okay. I'm sorry. I know you said this before, but you gave me some way of -- of tagging half-lives to chlorine atoms.

Can you just repeat that? I mean, what dictates that?

A The more -- the more chlorine atoms, the -- the longer the half-life. And the slower it is excreted into the body in general -- and that holds for the furans and the PCBs as well.

Q Have you done any investigations of exposures of the Penco Hosiery plant in Grenada?

A Exposure estimate?

Q Um-hmm.

A No. My understanding is that the other potential sources of pollution in the neighborhood were examined by other experts. I did not specifically focus on other sources.

The only other source that has been mentioned to me as being a source of some contamination, at least

in the ground water is the Heat Craft plant. I have not heard any discussion about the hosiery plant.

Q Yeah. I am not talking about the hosiery plant being in the neighborhood. I believe the -- Sherrie Barnes and some other plaintiffs in this case actually worked in the hosiery plant.

A Yes, I believe that's correct.

Q Did you do any investigation of what they might have been exposed to at work?

A Well, the history that was taken by Dr. Sawyer specifically indicated that they did not have any chemicals that they were aware of at that plant while they were working there. I mean, that was the family given history for Sherrie Barnes.

Q Sure. And you were relying on Dr. Sawyer for that piece of your analysis to the extent that is even part of your analysis; is that correct?

A Yes. In terms of looking for confounders or additional risk factors, that did not appear to be one.

From what I know about the hosiery manufacturing business, it is mainly a garment-type operation where they are making garments of clothing. And the hazards that might occur in such a setting would be lint if it was cotton that they were using. It could be some exposure.

I have studied garment workers in the past, and usually there is no measurable effect in work in the garment manufacturing in terms of lung disease or cancer risk or any of these other issues. So as far as I know, there would be no confounding from that source.

Q Well, so if they were actually making nylon hosiery, do you think there might be any exposures to any -- whatever chemical is going in to making nylon?

A If you were at a hosiery plant, you are probably not making nylon. You are buying nylon from a chemical company that makes the nylon. You are not making --

(Telephonic interruption.)

THE WITNESS: -- nylon material that would be present --

THE REPORTER: I'm sorry.

THE WITNESS: -- in the setting there.

THE REPORTER: Is that my phone or your phone?

THE WITNESS: No. It was noisy. I think that was the first couple of notes of Unsolved Mysteries.

THE REPORTER: It's mine.

(Whereupon, the record was read as follows:

"Q Well, so if they were Actually making nylon hosiery, do

You think there might be any Exposures to any -- whatever Chemical is going in to making Nylon?")

THE WITNESS: Nylon manufacturing is not done by very many companies. In fact, as I understand it, nylon has become less and less popular. They use synthetic fibers, but they would make it into the cloth somewhere else. They would not be making nylon at a hosiery plant there.

BY MR. HOPP:

Q They would dye it somewhere else, die the fabric, or is that done at a hosiery plant?

A It depends. Most likely they would buy the fabric already dyed in various colors. That is the normal clothing manufacturing practice.

They don't -- if you go to a -- any of the garment manufacturing plants in Los Angeles, for example, they don't do dying in those plants. They buy the fabric dyed somewhere else.

Q Let's talk specifically about Sherrie Barnes.

What was her body mass index at the time that she contracted cancer?

A Well, let's see. We need to look at the file. I think she was 190 pounds, but I forget her height.

Q While you are looking, how does one calculate body mass index?

A Let's see. I think it is the weight in kilograms divided by the height in meters squared. Something like that.

Q Okay.

A She was diagnosed June 15, 1997. I have to find her weight to see if anybody bothered to write it down. Let's see. She was 202 pounds on June 20th, 1997 and her height was 66 inches.

Q So based on that, one could calculate her body mass index?

A Yes. She had -- 2.06 was her height in meters squared, in kilograms was about 90. So it would be -- it was around 40, 45.

Q 40 to 45 was her body mass index?

A That would be in that range.

Q Did her body mass index change much during her adult life?

A I don't have that information. I mean, I think that she lost some weight. Let's see.

No, she didn't lose any weight. Between the time she was diagnosed and the time that she died, she stayed around -- around the 200-pound range. I think the last weight that was recorded was 190.

Q She was in her mid to late 30's when she passed away?

A 35, I believe when she died.

Q She died in '98?

A She died in September of '98.

Q She was born in September of '62?

A That's right. So she was 36, just -- just 36 or maybe she was a few days shy of 36 when she passed away. She was still in the 35th year.

Q What was her age at menarche?

A Menarche. Menarche sometimes said -- I don't know if that information was obtained. It is on my questionnaire. So let me try to find that. The daughter may not have known.

12. It was written down. So she had menarche at age 12.

Q Would that be considered early?

A No, it is not. It is smack dab in the middle of normal range.

Q What was her age at her first full-term pregnancy?

A I guess, I have to find out the age of her daughter and figure that out.

Q Well, she had the one daughter, Kenesha. Was that her only one child? Do you know?

A Well, I have to find my report.
Q Just for your reference, Doctor, I am handing you Exhibit 34 and 35 which were previously marked.
This is your narrative report and then the questionnaire from Kenesha Barnes.
A Okay. 22 was the daughter's age, so 22 from 35, 23. So she was 23 when she had her daughter.
Q That is when she had Kenesha. Do you know whether she had a full-term pregnancy before Kenesha?
A Well, that's a possibility. Number of pregnancies: One. Number of live births: One. So she only had one pregnancy and one birth.
Q Do you know how many months she lactated?
A No. I don't think we obtained the history as to whether she breast fed or not.
Q Was she in menopause at the time that she was diagnosed with breast cancer?
A She stopped menstruating when they started the chemotherapy, not before. She stopped menstruating at 34 with the chemo.
Q So we assume that she was not naturally in menopause at that time?
A Correct.
Q I think we established it last time, just to double check today, it is accurate to say that we do not

know whether she had ever used hormonal contraceptives?

A No. The only medicine she took was high blood pressure medicine before the -- before the diagnosis was made.

Q Did her questionnaire specifically ask whether she had ever used hormonal contraceptives?

A No. It simply said, what medicines were you taking, and we didn't ask -- I don't remember asking specifically about her form of contraception prior to the -- in the histories of Sawyer, myself, and Wolfson. I did not see any mention of contraceptive use.

Q And the history was given by her daughter. And so, I mean, was it reasonable to assume that her daughter may not have that information whether or not her mother used --

A Well, it is not just her daughter, but her mother and her sisters were interviewed and none of them were aware. It is most likely that she was on birth control pills, one of those family members would have known it.

Q But none of them mentioned it; correct?

A Correct. Thank you. And I believe it would have been asked by one or all of us who interviewed the family.

Q Okay. Do you know if she ever used hormone

replacement therapy for any purpose?

A As I said, there was no history of any other medication use.

Q Do you agree that a high body mass index is a risk factor for breast cancer?

A Well, I am trying to remember if that has been mentioned as a risk factor. I don't -- let me look in my -- I think one of these references does a review of the various risk factors. Elm Rich. Elm Rich is a review article on risk factors on cancer -- breast cancer.

Q Just for the purpose of the question, I would be happy if you could read off your screen what the risk factors that are mentioned in Elm Rich?

A I will do that when it comes up here. This is 1983, but I think it covers at least some of the more popular issues. They studied 1,185 women with breast cancer and compared the 3,227 controls. The risk of breast cancer increased with increasing age of first birth. This effect was not accounted for by parity.

Q What is parity?

A The number of pregnancies. An early age of first birth appeared to reduce the risk relative to no pregnancy; whereas, a late age first birth was associated with a higher risk. Relative risk decreased

with increasing obesity among premenopausal women. So in this study obesity was protective.

Q In premenopausal women?

A In premenopausal women, that is what I just read.

Q Okay.

A The risk was higher among those who were obese, but there was no evidence of a trend with increasing body mass index.

Q I'm sorry. That seemed to be contradictory, that last sentence?

A I am just reading from his abstract.

Q Okay.

A We can discuss it if you want, but anyway let me keep going.

Q Okay.

A Risk did not vary with the risk of abortion. Risk was lower among postmenopausal women than the premenopausal women of the same age. And increased with increasing age of menopause, bilateral oophorectomy -- let me spell that, o-o-p-h-o-r -- reduced the risk more than hysterectomy alone; the positive history of benign breast disease; a positive family history of breast cancer; Jewish religion; 12 or more years of education was each independently associated with increased breast

cancer.

Now, in terms of that contradiction about obesity, we need to go into more detail. Among premenopausal women, the relative risk estimate decreased as body mass index increased and the trend was that statistically significant. Among postmenopausal woman the opposite effect was evident relative to the BMI of under 30.

The relative risk estimate was 1.5, with confidence interval of 1.2, 1.9, and that was for postmenopausal woman and that was the body mass index was under 30, the relative risk was higher.

Q In postmenopausal?

A Postmenopausal does not apply to our patient who was not postmenopausal.

With a body mass index over 30, there was no evidence with a trend for a relative risk of increased cross-categories of increasing body mass.

Q So over 30, there is no increasing trend in both pre and post or --

A Correct. So I think the answer is -- and I think this is one of the biggest studies of that issue. It does not appear that obesity is a major factor.

Let's put it that way. It may contribute in some way, but it says among premenopausal, which is our

group, the relative risk decreased as the body mass increased.

Q Okay. Do you agree that age at menarche -- menarche is a risk factor?

A Well, it has been mentioned. This particular paper -- let's see. Where does it talk about it?

Risk according to menarche -- menarche, those that have menarche under 12, the relative risk was 1.4, but it wasn't statistically significant. The relative risk if they had menarche at the age of 11 to 12 was 2.1.

Q Which is increased; right?

A Which has increased.

Q You are looking for a relative risk number of one for it to be normal; correct?

A Correct, but none of these are one. It is kind of interesting. One has to wonder where -- 13 to 14 the relative risk is two, but above 15 it is one, so -- but it is rare to be over one.

Of the total population, there is only 200 people out of 5,000 that had menarche over 15. So that is not statistically significant because it is stayed with small numbers. So none of these ages at menarche in premenopausal women were at one. They were all elevated.

Q All right.

A So that is interesting and it holds up for postmenopausal as well. The values are all 1 at postmenopausal. So it appears to be that all of the premenopausal women seemed to have a higher risk no matter whether they were before 11, 11 to 12, 13 to 14, the only ones that were not elevated were above 15.

Q Do you agree that age at the first full-term pregnancy is a risk factor of breast cancer?

A Well, that is what this made -- this one is all about. It says, "Age of first birth," you know, that is the other table here.

Q All right.

A And what it says is that if they have the baby, first baby, under the age of 20, the risks are reduced.

If they have the baby between 20 and 24, the risk is at one across the board. One and 1.6 and 1.9, similar to under 20.

If it is between 25 and 29, the risk goes up overall, and it is statistically significant. So having the first baby after 25 raised the breast cancer to 1.7, 70 percent increase; and over 30, it is about the same. 1.8 is the relative risk.

Q Do you think that age -- strike that.

So you agree that months of lactation is a risk

factor for breast cancer?

A You know what, they did not study that in this paper, and I have never heard about it being a risk factor. So I don't know what the answer to that is.

Q Now, the issue of smoking and breast cancer, is it accurate to say that that remains somewhat controversial?

A Well, there is evidence. A number of studies have found a link. And, in fact, there is even a study that found a link with secondhand smoke. So I think the evidence is building that cigarette smoke contributes to the risk of breast cancer.

Q All right. Well, do you agree that race is a risk factor for breast cancer?

A Yes. It is more common in white women than black women. So it is -- appears to be, as I had said here, that women who have higher education levels and other studies have shown that are more common in upper middle class women than in poor blacks.

Q Does anybody have any notion for what the basis for that is? Why would women who have more education have a higher risk factor?

A Higher incomes probably eat more fish and eat more PCBs and dioxins.

Q More fatty foods?

A No. More fish.
Q More fish.
A Because the biggest source at this point for all of the halogenated persistent organic pollutants is fish.
Q There is a series of studies done in the '80's, I believe, in Michigan having to do with fish consumption and dioxin. Have you reviewed those? Do you know which studies I am talking about?
A There was one study of Michigan, studying women that ate more fish that was locally caught, wild fish, not -- not commercial fish. They were studying the women who were eating the PCB laden fish --
(Telephonic interruption.)
THE WITNESS: -- out of the sport fishing activities.
BY MR. HOPP:
Q Out of the Great Lakes?
A Out of the Great Lakes.
And there was -- what I remember is that they found elevated values. I don't remember if they studied any of the disease outcomes. I have to rereview that.
Q When you say that the fish consumption is a major source of dioxin exposure, are you talking about sport fish or commercially grade fish?

A Well, right now, it is both, especially farm-raised salmon is quite high in dioxin and dioxin-like compounds.

And there is -- also depending on the water they come from -- sport fish, like the Santa Monica Bay here, the fish are very high in PCB and dioxin. It used to be signs on, it appears, "Do not eat the fish."

Somehow the health department exhibited the wisdom to come and had taken those signs down; but they are still here, like the Santa Monica Pier here, there is a little, tiny sign that you can barely read that says, "Don't eat the fish."

Q Sure. We got the same problem in Lake Michigan. Depending on whether you were pregnant or of a certain age and that had to do with --

A Mercury. Mercury. That was mercury. Mercury is another contaminate in fish and it is a major issue for pregnant women.

Because if you do eat three fish meals a week, you have a risk to have enough high level to impact your offspring.

Q Do you agree that the use of hormonal contraceptives is a risk factor for breast cancer?

A I believe it is, yes.

Q Do you agree that the use of hormone

replacement therapy is a risk factor for breast cancer?

A Yes, it is.

Q Are cancer rates in Mississippi the highest in the nation?

A I have got a paper here by me that talks about --

Q By who?

A By me. I think the guy's name is Mitra. He is a Mississippi research guy. Let me see what he says.

Q Which list is it in?

A The breast cancer list. Breast cancer in Mississippi. Anyways, he went to each county and looked at county incidents versus state incidents. Incident rates of female breast cancer in Mississippi in 82 counties is 61.2 per 100,000 in 1996.

Q That's state-wide?

A That's state-wide. Whites were higher by a big factor, more so than blacks or non-whites, which is mainly blacks.

And there are certain high risk counties in Mississippi that correlated with pollution. Six counties had rates 40 percent higher than the state rate. And those six counties are listed here. He did not include Grenada County.

Q Where did Grenada County stack up in terms --

as part of the state?

A It is part of the lower ones. It is the white state, which means the rate is significantly lower than the state rate, and it has not been increasing.

Q You said, "It is a white state." You mean a white county?

A A white county, correct.

MR. PRUDHOMME: I think he is talking about the shaded areas.

THE WITNESS: The shaded -- the dark ones are the counties with the highest rates. They have a 40 percent or more increase in breast cancer and the white states have shown no increase.

BY MR. HOPP:

Q Again, you mean white counties? You keep saying, "States."

A I mean counties.

Q Okay.

A I'm sorry. And the maximum air pollution levels correlated with the rates.

In other words, if the county had more pollution based upon its toxic release inventory, it had higher rates. Grenada, again, was not one of the states -- one of the counties with the higher rate.

Q So to summarize then, based on the paper you

are reading -- which, again, can you give me the author's name?

A Mitra, M-i-t-r-a.

Q Okay. Based on the Mitra paper, whites in Mississippi have a higher risk of breast cancer than blacks; correct?

A Yes. Yes. The white rate is 1.8 or 61.4 per 100,000. The non-white is 52.3 per 100,000.

Q Does the Mitra paper discuss at all where Mississippi ranks in terms of the 50 states as far as cancer incidents is concerned?

A I thought he had mentioned that here. Let me go down to the Discussion and see.

No, I -- I don't -- I don't think he mentions that whether they are lower than the rest of the country or not.

Q Are you aware of any reference that discusses where Mississippi ranks among the 50 states in terms of cancer incidents at all sites?

A I did not remember Mississippi being the highest state rate for cancer overall. I remember that Louisiana and New Jersey had the higher rates than Mississippi, but that's just my recollection. I didn't research that question.

Q Do you have -- do you have any recollection of

any studies indicating where Mississippi ranks among the 50 states for the incidents of breast cancer specifically?

A No, I don't.

Q I want to skip back to the New York City firefighters for just a few questions.

Yesterday I was searching for the name of a drug that I thought one of the papers you mentioned that you administered. The drug was called cholestyramine, c-h-o-l-e-s-t-y-r-a-m-i-n-e.

Did you or someone else administer cholestyramine to the New York City firefighters?

A No, it wasn't part of their treatment.

Q Did an institutional review board approve the firefighter research paper -- firefighter research project?

A Yes.

Q Which one?

A The one that we maintain in our institution, our own in-house IRB.

Q Which institution are you talking about?

A Well, within the -- within my -- my -- my practice, I have a group of people that we sit down and review it. And it is our own internal review board.

Q So that is the internal review board at James

Dahlgren Medical?

A Correct.

Q And who sits on the IRB at James Dahlgren?

A Ren Schmidt, Pam Anderson, Harpeet Tarkar, and myself.

Q You said Ren Schmidt?

A Um-hmm.

Q Is the first name R-e-n?

A Reynold. It is R-e-y-n-o-l-d.

Q And Pam Anderson, Harpeet Tarkar, and yourself?

A Correct.

Q And I know you are an M.D. I know Harpeet Tarkar is not an M.D. Is Pam Anderson an M.D.?

A No. She is a Ph.D.

Q A Ph.D. in what?

A Epidemiology.

Q And what is Ren Schmidt's professional qualifications?

A He is an M.D. and Harpeet Tarkar has a master's in epidemiology.

Q Do you have any formal report from the IRB at James Dahlgren Medical authorizing or approving the New York City firefighter program?

A Yeah, we have one somewhere. I'm not sure where it is at this point, but yes, we do.

Q But you document the work of your IRB approved study?

A That's correct.

Q Is that a standard procedure; that is, for a doctor who is going to head a study to sit on the IRB which approves the study?

A It happens sometimes, yes.

Q Is it common?

A I don't know how common. I never studied it.

Q For the New York City firefighter project, did any of the firefighters receive a placebo treatment or some sort of sham treatment to check placebo effect?

A No. I think I mentioned yesterday, we tried to figure out if it was some point. There is no way you can have a placebo sauna, except sit in a room with no heat and with --

Q Maybe not hot enough. I don't know.

A Well, anyway, I think the main way to do it is to match them with patients who are similarly situated and see what happens to them with no treatment, no activity.

And we've actually followed how several dozen of these firemen, who didn't get treated, and they have not gotten any better.

Q Okay. I have got the recent paper that you

handed me entitled Persistent Organic Pollutants in 9/11 Rescue Workers: Reduction Following Detoxification. And this is a follow-up of the paper that we marked at the session; is that right?

A This is the paper that we presented at the meeting. The other was an abstract for the purpose of securing a position to make the presentation. This is what we presented in the meeting.

MR. HOPP: Let's mark this as an exhibit.

(Defendants' Exhibit 127 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Dr. Dahlgren, I am handing you what we have marked as deposition Exhibit No. 127, and this is the Reduction of Detoxification paper.

And I appreciate your providing me with a copy of it this morning. I have not finished reading it, but does this paper compare the firefighters who received the detoxification treatment with one or more firefighters who did not?

A No, we didn't put any data in there. We didn't have any measurements of other firefighters. I am just indicating to you that we have followed a group of these fellows, who didn't get treated, and they continued to be symptomatic, continued to require medication,

continued to be unwell. So just the passage of time doesn't -- doesn't explain the improvement.

Q Have you taken blood level measurements from the group of firefighters that you are following who did not receive the detoxification treatment?

A No, I didn't. The fire department did take PCB levels of -- on 1200 firemen, I believe, maybe more, and found elevated values in some of them. We haven't been given -- given that data. We just been told about it.

Q Okay. And you said the fire department took blood levels, is that recently? That is several years postclean-up, or was that --

A No. That was 6 to 12 months after clean-up.

Q And are there more recent blood level measurements in the people that did not receive the detoxification treatment?

A I do not know of any follow-up on those people in terms of measurements.

Q So the following --

A I know about the clinical status, but in terms of measurements of PCB's, I don't think that has been done.

Q By "clinical status," you mean their symptoms?

A Correct.

Q Do you sit on a medical advisory board for the

New York Rescue Workers Detoxification Project?

A Yes.

Q I just want to go through some other names to ask you whether these people also served on the board.

Does Mary Cecchini, C-h -- Cecchini, C-e --

A Cecchini.

Q There we go. C-e-c-c-h-i-n-i, does she also sit on the board?

A Don't know.

Q Does Bob Graves also sit on the board?

A Don't know.

Q Does Kathleen Kerr, K-e-r-r, also sit on the board?

A Don't know.

Q Does Keith Miller also sit on the board?

A Don't know.

Q Does Ernest Pecoraro, P-e-c-o-r-a-r-o, also sit on the board?

A I don't know.

Q How about Rita Weinberg, W-e-i-n-b-e-r-g?

A I don't know.

Q Jim Woodworth also sits on the board?

A I don't know.

Q Do you know who any of your other fellow board members are?

A Dave Root is the only one I know.
Q Rude?
A Root, R-o-o-t.
Q Do you have meetings with this advisory board?
A I believe we had one meeting that I remember with New York City, maybe a year and a half ago. It does not have regular meetings, obviously.
Q Do you know Mary Cecchini?
A Yes.
Q Have you worked with her in the past?
A Yes.
Q On what?
A On this project on analyzing the data for the firefighters.
Q Have you worked with Bob Graves on this project?
A No.
Q Do you know Bob Graves?
A No.
Q Have you worked with Kathleen Kerr on this project?
A No.
Q Do you know Kathleen Kerr?
A No.
Q Do you know Keith Miller?

A Yes.
Q Have you worked with Keith -- Keith Miller on this project?
A No.
Q And what is Mr. Miller's sort of professional background, if you know?
A He is a businessman. His job is -- used to be to administer the clinic that did the detoxification procedure for Dr. Root's practice in Sacramento.
He is also the head of a foundation called The Foundation for the Advancement of Science and Education here in Los Angeles.
Q Is that Advancement in Education?
A And Education.
Q And Education. Dr. Root's practice is at Health Med Sacramento?
A Yes. That is his clinic where he does the detoxification in Sacramento.
Q And Ernest Pecoraro, do you know Ernest Pecoraro?
A No.
Q Do you know a Cal Smith?
A No.
Q Now, you do know April McNight; right?
A Yes. She is the doctor who runs the downtown

medical clinic where the detoxification has been done for the last two plus years.

Q Do you know Rita Weinberg?

A Yes.

Q Have you worked with Rita Weinberg on anything other than the detoxification project?

A No, she doesn't really work on it anyway. She is just one of the friends of Keith Miller who frequently accompanies him on his enterprise or visits to New York.

Q Do you know Jim Woodworth?

A Yes. He is the administrator of the downtown medical clinic.

Q Has he also -- he also worked for Health Med in Sacramento?

A Yes. He used to run that clinic.

Q Did you work with Jim Woodworth on anything other than the detoxification project?

A No.

Q Are you aware of any studies that correlate PAH and dioxin exposure with breast cancer strains that are resistent to treatment?

A I have not seen any data on distinguishing cancers that are resistent to therapy versus cancers that are more responsive to therapy.

Q Are you aware of any studies that correlate PAH or dioxin exposure with breast cancer strains that are likely to metastasize?

A I have never seen studies that differentiate in that way.

Q All right. Can you quantitate Sherrie Barnes' risk for breast cancer using the Gail Model? G-a-i-l.

A No, I don't know how to do that.

Q Do you know what the Gail Model is?

A No.

Q Do you think that Sherrie Barnes' mother Mary Barnes is at increase risk for breast cancer?

MR. PRUDHOMME: At present?

MR. HOPP: At present.

THE WITNESS: Oh, gosh, I don't know. Let's see. I don't remember what her mother's history is.

BY MR. HOPP:

Q Well, if -- if I can refresh you, I believe that Sherrie Barnes' mother testified that she moved into the house in Carver Circle in 1961 or so, just before Sherrie was born and she lives there today.

A And she is now in her 60's?

Q I think so. I'm not quite sure how old she is. Probably in her 50's or 60's.

A Well, she had to be in her 60's and moved into

the house and had the baby at '61. She had to be at least 18.

MR. WINTERS: I thought she was 69 or 70, in that range.

THE WITNESS: The risk drops off as you get older, you pass certain milestones in age, but it is usually a little older than that. She is probably still at risk for breast cancer.

BY MR. HOPP:

Q Do you think Kenesha Barnes is at increase risk for breast cancer?

A Yes.

Q Based on environmental exposure?

A Based on environmental exposures. And the sister, the two sisters, and if there is an interaction, and I believe there is the environmental factor and host factors, then they would be at increase risk based on environmental exposure plus the history.

Q You believe then -- just to be clear then, you believe that Kenesha Barnes is at an -- you believe that Kenesha Barnes is at an increased risk for breast cancer based in part on the fact that her mother and her maternal aunt had breast cancer?

A Yes.

Q Do you believe that Kenesha Barnes -- strike

that.

Do you believe that Sherrie Barnes' sisters are at an increased risk for breast cancer?

A Yes. I think the sisters -- I should have found out, I guess, but I don't know where in the birth order Sherrie Barnes is. And what is her name? Kay Hobbs. I don't know if the sisters are older or younger. I just don't remember, but I think they would be at increased risk probably because of exposure.

Probably exposure. I have to confirm that, but if they were, indeed, exposed in the Carver Circle home, they would also be at increased risk.

Q Do you think that they, the sisters of Sherrie Barnes and Kay Hobbs, also has a host factor that would increase their risk?

A Yes.

Q Are you aware of any studies indicating that TCDD is chemoprotective for breast cancer?

A Yes.

Q Are you aware of any studies indicating that PCBs -- certain particular PCB congeners are chemoprotective for breast cancer?

A No, I am not aware of that. I have not reviewed that particular question but CIIT, C-I-I-T, composed a paper, did some rat studies that showed that

TCDD is reduced, and prevents the breast cancers, but reduces the numbers and prolongs the time that it took for the PAH that they used to induce the man-making cancer to occur.

So it was a study that indicated that the TCDD somehow had a -- what they thought was an anti-estrogen effect. And that that allowed it to then reduce the potency of the, you know, chemical that was used to induce the brain cancer -- breast cancer in an animal study.

Q Would you characterize that study as junk science?

A No. It is an interesting study.

Q A lot of what we talked about earlier today with respect to breast cancers and risk factors sort of the common thread running through a lot of those risk factors is estrogen; is that right?

A Yes. It is felt that breast cancer is at least one of the mechanisms and one of the factors is estrogen. Some kind of interaction with other factors, obviously, because estrogen is a normal necessity for normal development, but there could be some kind of derangements.

So maybe with higher levels which is why birth control pills, hormone replacement, are suspected to be

increasing the risk because you have an increased amount of estrogen that somehow creates an imbalance.

Q And something that is an anti-estrogen is, at least in theory, are potentially chemoprotective for breast cancers?

A Yes. And there hasn't been any follow-up that I am aware of that looked at that question, but the other side of the coin is, that a study was done also in rats where they exposed the fetus by exposing the mother rat to TCDD in a single dose during pregnancy -- and early on in the pregnancy, and then looked at the breast cancer risk in that fetus when it grew -- grew up.

And interestingly enough, there was an increase in risk of breast cancer in that setting. So the timing of the TCDD exposure is important in terms of breast cancer risk.

Q And I think you mentioned that study yesterday.

Is that contained within your bibliography?

A Yes.

Q Can you tell me the name of that particular study?

A Let me check to see in here what I thought it was. At least one of the papers addresses this question is the Vorder Strasse, V-o-r-d-e-r, S-t-r-a-s-s-e, paper and the other -- let me look at the reference list here.

Q Are you aware of other studies that discusses the issue of administering TCDD to rats during pregnancy and following their offspring for incidents of breast cancer?

A The other paper that I was talking about was Birnbaum, B-i-r-n-b-a-u-m, 2003. "Prenatal exposure to natural and synthetic estrogens is associated with increases in breast and vaginal tumors in humans as well as uterine tumors in animals. And then they talk about these issues.

Q This is Birnbaum and Fenton?

A Correct.

Q 2003?

A Correct.

Q The title is Cancer and Developmental Exposure to Endocrine Disruptors?

A That's right.

Q National Health and Environmental Effects Research Lab?

A That's right. It is a review paper, and she talks about these various issues that I just mentioned.

See, if I can find the other section, she talks about dioxins. The term dioxins is used for members of the PHAHS, that would be polyhalogenated aromatic hydrocarbons, that are structurally related and have

similar halogen substitution patterns are persistent and bioaccumulative, and have a common spectrum of biological responses mediated via binding to a specific high-affinity cellular protein, the aryl hydrocarbon receptor.

The prototype chemical for this class of dioxin or TCDD, and it goes on to discuss its developmental toxicity.

Let me see if I can find it.

Q 128.

(Defendants' Exhibit 128 was marked for identification by the court reporter.)

THE WITNESS: Let's see if she says that.

BY MR. HOPP:

Q Are you finished looking for it?

A No. I am looking at this prenatal -- this whole section called Prenatal Endocrine Destruction and Mammary Tumors. It is on -- you got the page in front of you? It is on Page 392.

Q And just for the record, we have marked the review paper as deposition Exhibit 128.

A Yes. And here is Brown.

Q So she is citing a paper by someone named Brown?

A Yeah. Brown is the other paper which is the

one that I am looking for. Brown '98. Has the prenatal TCDD exposure.

This was the one I was specifically referring to. The rats were gavaged with one microgram of TCDD per kilogram on day 15 postconception.

Q So Brown is the -- the rat study?

A The one that I was specifically referring to. They then looked at the response of those rats to a mammary carcinogen, which I thought was mentioned here, but let's see if I can find it.

Prenatal TCDD treatment increased total proliferative compartments in the terminal endbuds in 50 day-old rats. Prenatal TCDD resulting in an increased number of mammary adrenal carcinomas in rats.

Let's see see what they used for the induction. DMBA, dimethylbenz[a]anthracene.

Q Is that a PAH?

A It's a PAH.

Q So it is not a dioxin?

A No. It is a PAH. DMBA, which is a PAH and anthracene. So that this was the specific one, but there was -- Birnbaum talks about some others.

Q All right. I found actually Birnbaum Vonder Strasse and Brown. I would like to dig through them one at a time.

Birnbaum, first of all, is deposition Exhibit -- Birnbaum is deposition Exhibit 128 and Birnbaum is a review paper; right?

A Birnbaum is a review paper.

Q So it is not an -- it is not an original research project, but rather a summary of other people's published work; correct?

A Yes.

Q And Birnbaum, as you have stated, is looking at prenatal exposure to TCDD as a risk factor for breast cancer; correct?

A Yes.

Q And one of the questions that Birnbaum asks at the end is -- well, let me read it to you.

She talks about other studies and says,

"These particular studies have
Measured the levels of exposures
Of these chemicals in adult women
Who develop breast cancer. Could
We be trying to correlate exposure
And effect at the wrong time?"
If it is early or prenatal life
Stage exposure that is critical to
disease susceptiblity, why are we
measuring the environmental

Chemical in people once they have developed breast cancer? The Critical exposure may have occurred Much earlier."

Those are the last words in the Birnbaum article.

A Sure.

Q What is your response to those questions?

A I think that is exactly right. There is nothing wrong -- I think it is an extremely important point.

Q So do you think that -- that Sherrie Barnes' risk of breast cancer may have been influenced by her prenatal exposures?

A Yes.

Q Does --

A For example, her other sisters might have been in utero elsewhere prior to '61. That is one of the questions I don't know the answer to, which in Kay Hobbs and Sherrie maybe would be the ones that were in utero in the Carver Circle area.

Q Okay. And she may have been in utero before her mother moved to Carver?

A She may have been. '61, she was born -- '60 -- '62. It is likely she was pregnant when she was there.

She was nine months into '62 when she was born. So it is likely that she was conceived and the entire pregnancy was in Carver Circle that you just told me.

Q Right. And I may be incorrect. We will have to check that, but either way, whenever she moved into --

A Whenever she moved in is when her exposure started. That does not mean that it has to be prenatal exposure.

I am just agreeing that Dr. Birnbaum it is probably an important issue and needs more attention. We have to look at these early developments.

In fact, another major issue, which she does not address in any detail -- I don't think Dr. Birnbaum has not had a big focus on this, but I have been very interested in it, and that is so-called male mediated developmental toxicity.

That is sperm exposure to these kind of chemicals, altering the sperm and then increasing the risk of cancer in the offspring.

There are a number of studies, not with dioxins or PAHs, but with other chemicals like arylamine is the most common one.

If the father is exposed at the time of conception, that baby, in this case rat, is, more likely

to develop certain cancers.

Q And do you think that may be an issue with Sherrie Barnes given that her father had breast cancer or may have had cancer?

A Yes. That sees to be some question about cancer. Again, that could be an issue. Could be an issue.

Q Now, did Birnbaum's article -- if I understand it correctly, was more asking questions than answering them? It was a hypothesis-generating type of paper?

A Well, she also cited a number of studies that indicated that -- you know, I think quite clearly the studies that she cited weren't just speculation. They were data. And they weren't just hypothesis. They were data.

And she is generalizing from this by pulling together -- you understand that Linda is a policy walker. She is a person that tries to get people to do research in certain areas and she finds money for them.

So what she is saying by this paper to the community, hey, guys, I will give you some money to study this question.

Q Linda Birnbaum is with the Environmental Protection Agency?

A Correct.

Q She is not an epidemiologist?
A No. She is an epidemiologist.
Q And her job is more policy than research?
A Well, she does some of her own research but very little. She mainly reads and studies and funds and gets people to do things, rather than spending a lot of time herself in the lab.
Q Does the Birnbaum paper give relative risk data for breast cancer?
A Relative risk?
Q Right. Does she calculate relative risk?
A I don't see any relative risks in here.
Q It is not a case control study or a cohort study; is it?
A It's not even a review paper of that data. This is mainly a mechanism paper we are talking about, what is it that causes, among other things, breast cancer.
Q She hasn't even isolated that mechanism. She identified papers which look at that; correct? She is not committing to -- Ms. Birnbaum is not coming to any firm conclusions on plea condition subpoenas in her paper; does she?
A She does talk about endocrine disruption, the Ah receptor, and the anti-estrogen effect that we were

discussing a minute ago.

And she -- she even gives this reference that we talked about. I believe she gives a reference about the anti-estrogen effect.

Anyway, she talks about the Ah receptor noting that -- there it is on Page 392. Again, she states,

"The Ah receptor, which is required for dioxin effects, is present during organogenesis in most tissues. It continues to be expressed in the mammary gland of the pubescent rodents and is localized in the mammary ducts and developing lobules. In addition, these authors demonstrated that mice in which the Ah receptor has been eliminated display decreased mammary gland size and supressed lobule development, suggesting a critical role of the Ah receptor in normal and TCDD-exposed mammary gland development."

So, I believe, there are other paragraphs and little discussion about the Ah receptor.

Q But as we discussed yesterday, we don't know everything about the Ah receptor and how it induces

cancer; correct? There is still a lot of open questions about it?

A There are still questions, sure.

Q Now, did Birnbaum for the purpose of her paper, isolate the exposure at issue, or is she talking about all different types of endocrine disruptors?

A She talks about anthracene. She talks about other chemicals besides TCDD. She talks about nitrotoluene, DMBA, which we mentioned earlier and which is dimethylbenz[a]anthracene.

Q Another PAH?

A Another PAH, correct.

Q And PAHs are endocrine disruptors?

A Yes. They -- they can disrupt the function. Not so -- well, some of them have estrogenic effects, but they do stimulate the estrogen receptors. Some of them more so than others.

Q Are they weak endocrine disruptors? Did you say that yesterday?

A Yes.

Q They are more popularly known as being known as directly genotoxic?

A That mechanism is definitely present and one of the more potent. It -- it is the reason I think that PAHs are so toxic is because of their ability to bind to

DNA as we discussed yesterday.

Q Does Birnbaum document any of the exposure levels which are required to produce mammary tumors?

A She does not discuss that in this paper.

Q In your report at Page 116, you site Birnbaum's paper in the aid of the proposition that,

"We have much more work to do in order to clearly understand the mechanisms of action."

Do you stand by that statement?

A Yes.

Q And you believe that Birnbaum supports that statement?

A Yes. We need to know more. That's certainly true.

Q Let's talk about Vonder Strasse for a minute. This is Deposition Exhibit 129.

(Defendants' Exhibit 129 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Vonder Strasse looked at mammary gland differentiation; is that right?

A Yes.

Q And is it an in vitro study? Vonder Strasse -- oh, it is mice?

A Mice.
Q It is a mouse study?
A Mice, yes.
Q But it didn't directly study mammary gland cancer, correct, or breast cancer?
A No. It looked at a mammary gland alteration, which is thought to be indicative of the same type of disruption of the mammary gland and would lead to cancer, carcinogenic outcome.

The study was looked at in the different days of pregnancy and then looked at the mammary gland development, you know, after that.

They didn't go all the way -- let the animals grow up and expose them to the cancer-causing agent. It just shows the profound effect of TCDD on mammary development in utero.

Q And how the TCDD administered to the mice in Vonder Strasse?
A They gave them by gavage.
Q And gavage means to actually put a mixture of the toxic down the mouse's throat; is that right?
A Yes. They put a little tube down to the stomach and inject it. And they put in five micrograms per kilogram in peanut oil.
Q So it is a mouse-feeding study?

A Yes.
Q And this, the Vonder Strasse paper, does not give any relative risk data for breast cancer; does it?
A No. No, it is not the point of it.
Q And it studies TCDD in isolation; correct?
A Yes.
Q Let's look at Brown. Brown is deposition Exhibit 130.
MR. HOPP: Did I give you Brown, Keith?
MR. PRUDHOMME: Yes.
(Defendants' Exhibit 130 was marked for identification by the court reporter.)
BY MR. HOPP:
Q All right. Well, Brown is another mouse study; is that right?
A Yes, it is another rat study.
Q It's rats this time. And this is, again, a rat-feeding study?
A I think we indicated it was gavage.
Q Yes. And just to be clear, gavage is the same thing that you mentioned before where they put it down the mouse's throat?
A That's right.
Q And it is one microgram per kilogram in this -- in the Brown study; correct?

A Yes, I believe that's right.
Q Administered a single time on day 15 postconception?
A Correct.
Q Now, on Brown in the Discussion section, Page 1625 states, "However, for every report of Dioxin being associated with breast cancer, there seems to be one that Finds no significant effect."
Do you agree with that statement?
A The statement speaks for itself. Yeah, there are some negative studies.
Q Well, looking at Brown's Conclusion, this is on Page 1628, Brown states: In humans, neither ecological data nor occupational studies, provide clear support for an association between organochlorine endocrine disruptor exposure in the occurrence of breast cancer."
Do you agree with that statement?
A No, I think that is wrong. I think that it's overly stated. I think the evidence is consistent and the other point that Brown makes is that it probably has to do with -- with the timing of the exposure.
Exposure to dioxin as we have been discussing earlier, in the -- in the adult may not increase the risk of breast cancer, which is one of the reasons you

get some of these -- you are looking at occupational studies; and that may not be the time when the breast is susceptible to the breast cancer induction.

There is a study in here that if you -- if you looked at adult exposure, if you look -- follow the hypothesis that has been generated here, you may not see an excess of breast cancer from dioxin alone. That is what is suggested by what we have been discussing this morning.

Q Let me just go on. In Brown's concluding paragraph, she says, "It is possible that

postnatal as opposed to prenatal exposure to TCDD may yield a different outcome, perhaps rendering a protective effect against mammary cancer."

That is what you were just talking about; right?

A Yes. That is what I said, estrogen effect. If you are exposed as an adult, it may be protective. That is a pretty amazing idea because you don't want to go around administering TCDD to people to prevent breast cancer; but, you know, conceivably based on what we discussed, it may not increase the risk.

It may be quite amazing if it actually decreased the risk because it would increase the risk of

other adverse outcomes. So I would not recommend it as therapy.

It is one of the reasons why you are seeing different outcomes and different studies just because of the timing. I think that is the point.

Q Sure. Brown went on to say, though,

"It is our intention to investigate a potential neonatal TCDD treatment to predispose for mammary cancer in the underlying molecular mechanism action for perinatal exposure to organochlorine."

Do you know if Brown ever did the following?

A Well, she is 98. I think the wheels of science move slowly.

Q Okay.

A I would suspect that she is working on that. She may not have gotten around to publishing it, but it's -- you know, usually these professors have a lot of things that they are trying to do. And it takes them a while to get things public.

Q Do you know Nadine Brown?

A No.

Q So you do not know whether she is working on it or whether she has moved on to something else?

A Correct. It is a question of funding.

Q Sure. And, again, the Brown paper isolates TCDD as the exposure; correct?

A Yes. And that's -- that is the way the research is usually done, as I discussed with you yesterday when you were talking about the individual dioxins. You know, you wouldn't have any reason to use any of the other dioxins. You would use this one.

Q Right. Does the Brown paper provide any indication as to what exposure level would be necessary to produce these effects in humans?

A Well, she used a very fairly low dose. I mean, a milligram per -- I'm sorry -- a microgram per kilogram. That's a pretty low dose.

In fact, I think, if I remember correctly, they didn't demonstrate the no effect level. It may well be that if you go down to lower doses -- and the effect may still be there.

I mean, this study wasn't intended to find out what the lowest threshold for this effect would be. You notice that -- what was it? The Vorder Strasser (sic).

Q Vonder Strasse.

A -- used five micrograms. This one they used --

Q I think it was one.

A -- one microgram, and they did it only once.

Q Yup.
A So that is a lot lower dose and they are still getting this effect. So it is really quite remarkable.
I mean, that is one of the reasons EPA's risk assessment that we were discussing yesterday has -- has become concerned because this is not a very -- not a very high dose.
Q I understand that Brown used a very low dose, but does Brown in her paper tend to extrapolate that low dose in rats to human effect?
A I have already stated that she did not do that. She did not attempt to -- to find a no effect level, and she did not attempt to extrapolate that to current human exposures.
MR. HOPP: Can we take five minutes for a comfort break, Dr. Dahlgren?
THE WITNESS: Yes.
(Brief recess.)
BY MR. HOPP:
Q I want to focus on the history that you took for Sherrie Barnes. When you talked to Kenesha Barnes and Mary Barnes and the other relatives, did you talk about Sherrie Barnes' diet at all?
A No, I did not ask about diet. I don't usually do that because I don't really know what to do with the

information when I collect it.

Q Do you know if Dr. Sawyer or Dr. Wolfson asked questions about diet?

A Dr. Sawyer did. Her diet history: Vegetable, primarily green beans and various greens. Ms. Barnes farm raised catfish approximately twice per month. Sherrie's brother William Jay caught fish from Bow Creek which was consumed by the family.

Sherrie consumed fish two or three times per month and occasionally bought fish at a local fish market or restaurant.

Q So any information on Sherrie Barnes' diet would come from Dr. Sawyer's report as opposed to your own data collection; is that correct?

A Yes. That is what I said. Like I said, I don't know what to do with that information because that is very similar to what most people would say.

They happen to be fish haters, which occasionally you run in to. People who never eat fish. And we know that that might increase the likelihood that you find PCB or mercury maybe at a lower level of someone that never ate fish; but still it is background.

Everybody in the South has PCB and mercury in their system.

Q Does Sherrie Barnes' fish consumption as

summarized on Dr. Sawyer's report strike you as abnormally high or abnormally low?

A No. I would say it is very typical of what most people would say.

I mentioned earlier a study that was done a couple years ago where they asked patients to eat more than -- who ate more than three fish meals a week to allow their blood to be sampled for mercury.

Among women child-bearing ages, about 20 percent, who had those three, had mercury levels that would be high enough where it would be harmful to the fetus if they were to have a pregnancy.

Q What are the toxic end points of mercury consumption when a woman is pregnant?

A Neurological effects in the baby.

Q Have there been any studies that you are aware of indicating that prenatal consumption of fish increases the baby's risk of breast cancer later in life?

A No studies.

Q Are you aware of any studies that talk about postnatal exposure to mercury being a perspective breast cancer?

A No.

Q Do you know whether Sherrie Barnes ever used

nonstick cookware containing Teflon?

A No, I didn't ask that question.

Q Now, you have been involved in litigation in something called C8; is that right?

A Yes.

Q What is C8?

A Perfluorooctanoic.

Q And is C8 a component of Teflon?

A Yes.

Q And what are the disease end points that are significant to C8 exposure?

A Cancer. Breast cancer and prostate cancer among others. Those were the most striking findings.

Q And the C8 case that -- that I'm aware of -- I'm not sure if this was the one that you are involved in or not.

The C8 case I'm aware of had to do with environmental contamination from C8 which had somehow allegedly got out of the factory.

A The water in the neighborhood got contaminated from the factory in West Virginia. Parker Springs, West Virginia.

Q Is there any literature that you are aware of cooking with Teflon-coated cookware increases a person's exposure to C8?

A No. No one knows how the C8 is getting into the blood of the general population, but it is there.

One possibility is Teflon cookware. But C8 is present in a number of other products. And there is probably more likely to be the root of exposure.

Q What are those products?

A Things like hydraulic fluids sometimes have C8 in them. And let me think. Gortex has it.

Q Gortex; is that fabric or rainwear?

A The big exposure is from Stain Master Carpet and other textile-treating chemicals that are used to make them -- make them. So they don't -- the stain does not stick on the fiber.

Q Okay.

A Stain Master Carpet is a DuPont brand and it -- they coat the entire fiber and the whole carpet is filled with this C8.

The Dutch Environmental Protective Agency did some studies and they showed that when you walk across the carpet that is treated with this stuff, you kick up molecules that are up in the air and so that may be one of the ways that they may be exposed. We just don't know yet. EPA is doing some studies on how it is getting into the people.

Q And does C8 -- strike that.

Does C8 give off gas from recently treated carpet?

A No. It is not volatile, but it comes off in particulates and there is some volumination. It is not totally lacking in volatility, but it is mainly the particulates that does it.

I mean, the air concentrations around the factory at one time in the past were quite high, and so there is some vapor that gets in the air. But no one has done measurements about how much is above the carpet. And the Dutch felt that it was mostly particulates exposure.

Q Now, one of the other exposures that you and Dr. Schecter studied recently is this fire retardant chemical product?

A PBDE, polybrominated diphenyl ether.

Q And that is something that scientists have recently found is in the environment in levels that no one ever suspected?

A Yeah. That was the main point of the paper -- the main point of the paper. That it is higher in the United States, particularly in breast milk than it is in Europe. That is because Europeans banned the stuff and which we have not yet done.

Q Do you know what the toxic end points are for

PBDE exposure?

A It is generally felt that it is going to be similar to dioxin. Limited animal studies suggest that they have the same cancer inducing, immune system damaging, neurological -- neurological damage, and endocrine disruption. So it has got all the similar toxic end points as dioxins and PCBs.

Q And do we know what the PBDE levels are in Mississippi, generally?

A We did them on these 29 people.

Q Okay. And?

A And they are included in that paper.

Q Well, let me back up. It is my understanding that the focus on PBDE is a new thing relatively, recent and people are discovering this as an issue?

A I would say it has been an issue in the last 10, 15 years.

Q And just to go back to your paper with Dr. Schecter -- I know we marked it here.

A I think it is here somewhere.

Q Here we go. What did you conclude? Deposition Exhibit 15, what did you conclude about the levels of PBDE in the blood of the 29 people from Mississippi as compared to 1973 serum levels?

A Well, we didn't have '73 PBDEs; did we?

Q Oh, is that the dioxin?
A Dioxin. We may have looked at it. This is breast milk, whole blood -- yeah. This is Mississippi and New York.
Q Okay.
A So these are the blood levels that we found.
Q What figure is that?
A Figure 3 and Table 4 is the 29 patients in this case.
Q And you found that their levels were high in comparison to somewhere else or --
A No. The levels were similar to what we found in New York. This is Table 3. New York is the firemen, and actually, the people in Mississippi were -- let's see.
Let's look at 99. Levels are similar between the firemen and the individuals in Mississippi. There is one person, 37-year-old female was high; 158 on PBDE, which is the one that is most abundant than anybody, but that particular person's is real high.
And who was that? That is an interesting question.
Q 37-year-old woman?
A Yeah.
Q Deposition Exhibit 39, is this the one that we

could not find from yesterday? I will give you my copy.

A Let's see if we can make a copy of this stupid thing. I wouldn't take your copy if we can avoid it.

I will use your copy. Okay. 37, in 2004, means that she was born in '67. So it was Lorethra Brown, '67.

Q And she had high PBDE levels?

A Yes.

Q Or high levels of one of the PBDEs?

A Yes. The one that you look at is 99. Among the fireman, the highest was 34. It was just one lady Lorethra Brown who did not have a big, high TEQ particularly.

Q A high TEQ per dioxin?

A Yes.

Q But she had a high --

A She had a high TCE level. I don't know why. It is a mystery.

Q Are there TEQs -- have TEQs been calculated -- strike that.

Have TEFs been calculated for various congeners for PBDE?

A I asked Dr. Schecter that question. I don't know if it is addressed here, but the short answer is no, but there has been -- somebody has at least raised

the possibility, but I have not seen any charts.

Q Is there a level of PBDE in blood which scientists believe gives rise to a health concern? How much do you need to make you sick?

A Well, let me read the -- let me read this sentence to you from the paper.

"Although there is no way at
Present to be certain of the
Nature and extent of the toxicity
Of PBDEs, which is especially of
Concern as PBDE body burned
Increases measure level, and toxic
equivalent factors and other pops, such
as dioxins, furans and PCBs decreasing
in human living in an industrialized
country." So there is no PCDF yet.

Q But PBDEs are going up while --

A That's right.

THE REPORTER: I'm sorry. I got mixed up with the --

THE WITNESS: Okay.

THE REPORTER: Hold on. Just give me the abbreviations. You got PDBE. What's the other one?

THE WITNESS: No. PBDE, polybrominated diphenyl ether. Yeah, it's alphabet soup.

BY MR. HOPP:

Q My question was PBDEs are going up while, I think, it was dioxins and PCBs are going down, and the answer to that question is "yes"; correct?

A That's correct. And the PBDEs were done on the '73 sampling and they were essentially nondetect for everything. So it wasn't present in '73 even, amazingly enough, but now it is present in significant quantities.

The pooled blood value totals showed a level of 61 parts per billion, whereas it was .77 parts per billion in 1973. And that serum, whole blood is 79; slightly more.

Q Okay. Now, we talked last time and a little bit today about dose calculations for Sherrie Barnes.

Did you do your own independent dose calculation for Sherrie Barnes' exposure to creosote and dioxin?

A No.

Q You relied on Dr. Sawyer for that; correct?

A Yeah, Dr. Sawyer. And Dr. Samara, also, I believe, gave information regarding that individual's exposure, but the main one is Dr. Sawyer.

Q Can you give me a dose of creosote or a dose of dioxin which you would consider to be a significant dose for the purpose of causing breast cancer or is that

something, again, that you would defer to Dr. Sawyer?

A Well, I think -- again, I would say similar to what Dr. Sawyer said, these people are at increased risk of cancer as a result of the exposure.

And specifically, one of the cancers to which they are at risk -- I mean, all of the people in this neighbor are at risk of breast cancer because of the nature of these chemicals that we alluded to in the last two days.

The nature of these chemicals being endocrine disruptors concentrating in the fatty tissue of the breast, specifically in the fairly active tissue, breast tissue.

Every tissue that these chemicals reach, it can increase the risk of the cancer in those tissues; but breast is particularly at risk because of its lipid nature and the lipid nature of these chemicals and because the metabolic activity and the sensitivity to estrogen which these chemicals mimic.

So for a variety of reasons, these chemicals we are talking about increase the risk. And as far as I know, there is no safe level of exposure to a carcinogen.

What we do with our quantitative risk activity is try to define the level which we consider to carry

with it a so-called acceptable level of risk, is a very low risk; but I don't know of any -- well, any evidence that there is a threshold for cancer effects.

So then the answer to your question is that any exposure is going to increase the risk. The higher the exposure, the higher the risk.

In these individuals, as Dr. Sawyer calculated in Sherrie Barnes in particular is significantly increased risk of breast cancer.

From his calculations, he calculated a dioxin dose, a PAH dose, naphthalene dose, creosote exposure levels, and so clearly, this -- this patient had a high risk.

Q Now, when I asked Dr. Sawyer questions about risk of breast cancer and dioxin exposure, for example, he answered by a reference to EPA slope factors for all cancers.

Are you aware of any science which isolates a dose of dioxin exposure which is significant for the purpose of causing breast cancer?

A Same answer. I don't think that -- none of the studies that I am aware of distinguishes between the different cancers.

Clearly, PAH and dioxins have both been shown to create cancers in animals and specifically, to create

mammary cancers.

I don't remember offhand that the slope factor was calculated from breast cancer in the occurrence and the lung cancers occurrence in the animals.

That is how slope factors are derived in animal studies with a single compound; and therefore, somewhat abstract and are mainly used for the comparison purposes so that we have some sense of the potency of this given chemical to cause a cancer.

As I said, like yesterday when you are in the real world, you are exposed to a variety of things and many of those things contribute to the risk, then the safe level of exposure of any one compound has to be reduced.

Q Just to be complete then, are you aware of any science which isolates a dose, the PAH, which is significant of causing breast cancer or is your answer the same?

A Yeah, my answer is the same. I don't -- I don't think there is any known threshold for cancer. So any exposure increases the risk. The higher the exposure, the higher the risk. And then it can occur at any tissue that the chemical is present.

And as I have stated, PAH concentrates in the breast has been shown to cause this type of cancer in

animal studies. And all of the things that we have discussed about dioxin apply to PAHs and so -- but in addition to it, its estrogenic quality and most important toxicity and its ability to disrupt DNA function; but it has been shown quite significantly to be present in patients with breast cancer.

PAH adducts is present in the breast tissue -- normal breast tissue adjacent to the tumor. And then the levels of these PAH adducts is so much higher in breast cancer patients than patients without breast cancer, showing quite clearly that it is probably a major contributing factor to occurrence of breast cancer.

Q Don't a lot of the recent studies on that -- on that subject in particular indicate that it is not clear whether the concentration of PAH, DNA adducts of breast cancer -- I'm sorry -- in breast tissue in people who have breast cancer is the cause of the breast cancer or a effect of the breast cancer?

A No. I think that the evidence is quite clear that what it means is that they have been exposed to more PAHs than other people. And therefore, that is why they are getting the breast cancer.

Now, there is -- there is susceptibility factors. Some patients are less able to repair the DNA

damage due to genetic differences. Some patients make more of the toxic intermediary due to genetic factor.

So there are susceptibility factors, but clearly, there is a dose effect as well when you are exposed to a higher dose of PAHs or dioxins, you are going to get more breast cancer.

Q All right. Let's -- let's go back a question.

In answer to one of my earlier questions, you mentioned the subject of threshold. Leaving thresholds aside, the EPA and other similar bodies have identified level of exposure to carcinogen including dioxin which they believe to be acceptable for policy reasons, if not scientific reasons; is that not correct?

A We -- they -- they come up with what they called cancer slope factors. And if you were exposed below that amount, their theory is that you will have an acceptable level of risk of developing the cancer.

Q And that applies whether the dose response curve for the carcinogen is linear or nonlinear. Even with a linear dose response curve, they isolate or identified an accept --

A It is a linear. It is a linear response curve that they are using to calculate the slope factor. And what they are doing is saying, okay, at this, you get one in a million or one in 100,000, or one in 10,000

depending on what date of the week, what they consider to be an acceptable level of risk.

Q Do you know what the acceptable level of whatever benchmark you want to use of exposure to dioxin is?

A Well, the EPA's level is a microgram per kilogram per day.

Q Do you know what the safe level of PAH exposure is for humans according to the EPA or any other benchmark?

A I don't think they have established a reference dose or they haven't expressed it quite the same way. The chronic oral level of acceptable PAH exposure, I don't recall from memory what it is, if they do have one.

Let me see. Maybe there is. Let me look at something. Maybe Sawyer has it here. What does he say about the number? No, he calculates from an EPA cancer potency factor of 730 micrograms per kilogram per day.

Q That is total PAH?

A It is a cancer potency factor. I think that's -- let me see if I could.

Q The question is micrograms per what -- microgram of what?

A Well, that is what I am going to look at. That

is PAH. Benzopyrene equivalent, just the carcinogenic PAHs. Yes, I think it is probably -- it may be benzopyrene. Let me see.

Yeah. I don't know how Dr. Sawyer got that EPA -- the dosage. Anyway, he has calculated the dosage. I have to ask him about where it came from.

Q If I were to ask you what level of PAH or dioxin exposure you would consider to be an insignificant increase of a person's risk of breast cancer, wouldn't your answer be referencing the case EPA slope factor and whatever their decision is is an acceptable level?

A Well, I don't know if -- sometimes the problem is the EPA plays games and they will come up with a slope factor of one in 100,000 and one in a million; and you ask them why? And they don't tell you.

But the usual, the oldest most common acceptable level of risk is one in a million.

Q So whatever -- anything under the one in a million risk is something that would be, in your view, an acceptable level of dioxin or PAH exposure?

A You know, if I was that one patient, I don't think that I would find it acceptable. And I have also indicated that, you know, there is no safe level of exposure that an individual patient can have.

This is the significant contributing factor. And if they hadn't had that exposure, they wouldn't have gotten the cancer.

So this is, you know, I mean -- just because it was, say, less than one in a million, I mean, you know, I -- I think that risk is certainly lower if your calculated risk is under one in a million.

Your question is do I accept that as sufficient? Excluded as the causative factor?

Well, I think we have to go on an individual case basis to see what is going on with that. For example, as I said earlier, if they are exposed to PAH at the one in a million risk, using this somewhat artificial construct; and they are at one in a million risk from the other chemical, both are going to be contributing.

And like I said before, the risk would have to be -- or the exposure -- acceptable exposure would have to be reduced to take into account the mixture exposure.

And in this case, we got dioxins. We've got PAHs. And we also have Benzene. Although, the dose is unclear. And then we have naphthalene.

Q Which is a PAH?

A Which is a PAH, but it has a separate slope factor because it is not included in the so-called

carcinogenic PAHs.

TEFs that are usually identified, but California has given a slope factor for cancer causation now. And there are, you know, animal studies to show that it does induce cancers. So it has to be added to our list.

Anyhow, just because the calculated PAH dose would be at one in a million, because of the circumstances in this case, it still may be contributing because of the synergistic additive and/or additive effect of the other exposures.

Q Let me ask you this: Do you think, leaving synergistic and additive effects aside, how low a dose would you consider to be too low -- strike that.

How low a dose would be too low for you to consider PAHs as a risk factor for breast cancer?

A I don't know the answer to that.

Q How low a dose would you -- strike that.

How low a dose would be too low for you to consider dioxin as a risk factor for breast cancer?

A Same answer, I don't know.

Q You indicated earlier that naphthalene has been shown in some animal studies to cause cancers; correct?

A Yes.

Q And forgive me if we covered this before, but

those animal studies were inhalation studies of rats?

A I don't remember whether it is inhalation or feeding, but it was rat studies, yes.

Q But do you know whether the cancer that was induced in the rats was nasal cancer?

A I don't remember. I would have to look at the article to see the answer to that question. I believe it may have been an inhalation study with nasal cancers, but I just don't remember from memory.

Q And you know that rats are obligate nose breathers; right?

A Yes, I do know that.

Q Are you familiar with the term organotrophotropism?

A Organotrophotropism, I think that has to something -- something to do with the tendency of a chemical to effect a certain organ. I think that is what organotrophotropism is.

Q In your clinical practice, have you ever prescribed a drug called Rifanpin, R-i-f-a-n-p-i-n?

A Many, many years ago, I think I wrote a couple of prescriptions for Rifanpin to treat some patient with tuberculosis.

Q Are you aware that it is an animal carcinogen?

A I have not remembered that, no. If it is, it

is not in my memory banks.

Q Do you remember giving any specific warnings when you prescribed Rifanpin regarding cancer risk?

A I don't remember.

Q Have you ever prescribed a drug called Isoniazid, I-s-o-n-i-a-z-i-d?

A I think that is misspelled.

Q I may mispronounce it, too. I-s-o-n-i-a-z-i-d. Does that sound like something else?

A I don't recall prescribing that.

Q Do you ever recall prescribing a drug called Clofibrate, C-l-o-f-i-b-r-a-t-e?

A Clofibrate is a cholesterol lowering agent. I've never prescribed it.

Q Have you ever prescribed Disulfiram, D-i-s-u-l-f-i-r-a-m?

A No. That's -- that's a drug to make -- to give to alcoholics to keep them from -- from alcoholics drinking because it makes them sick to drink.

Q All right. Have you ever prescribed Phenobarbital?

A I have prescribed that a couple of times, yeah.

Q Are you aware that that is an animal carcinogen?

A No, I was not aware that it was an animal

carcinogen.

Q Have you ever recommended -- strike that.

Acetaminophen used to be a prescriptive drug; is that right?

A You mean Tylenol?

Q Yeah.

A I didn't know that was ever a prescription drug.

Q Did you ever recommend people to take Acetaminophen?

A I definitely -- I always recommend patients never to take Tylenol or --

Q Why is that?

A Because of its liver toxicity. It is equivalent -- it killed more people last year than Vioxx and any of the rest of them. It is real a bad drug.

Q Is it a carcinogen -- an animal carcinogen?

A I don't know.

Q Have you ever prescribed a drug called Metronidazole? Let me spell it for you, M-e-t-r-o-n-i-d-a-z-o-l-e.

A Metronidazole?

Q Metronidazole.

A Yes, I have prescribed that.

Q What is it?

A It's an anti-parasite drug. It is used to treat things like Giardia and it is also used to treat anaerobic infections.

Q What is Giardia? Keith knows it.

A Intestinal parasites, very common.

Q Are you aware that it is an animal carcinogen?

A Yeah, I was aware of that.

Q When you prescribe it or when did you prescribe it, did you ever give warnings on that subject to the patients?

A No.

Q Have you ever prescribed a drug called -- and I need to spell this one, too --
S-u-l-f-i-s-o-x-a-z-o-l-e, Sulfisoxazole?

A I may have prescribed it once.

Q Do you know what it is?

A It is an antibiotic.

Q Do you know it was an animal carcinogen?

A No.

Q Have you ever prescribed Dapsone, D-a-p-s-o-n-e?

A No.

Q Have you ever prescribed Methimazole, M-e-t-h-i-m-a-z-o-l-e?

A No.

Q Have you ever prescribed Oxazepam, O-x-a-z-e-p-a-m?
A No.
Q Have you ever prescribed Furosemide? Furosemide, F-u-r-o-s-e-m-i-d-e.
A No -- well, I probably did when I was a resident.
Q Do you know what that is? What that drug is?
A Yes. It is a diuretic.
Q Are you aware it is an animal carcinogen?
A No.
Q How many cases of breast cancer are diagnosed in the U.S. each year?
A 160,000, in that range.
Q Do you know how many cases are attributable to creosote exposure?
A No.
Q Do you know how many of those cases are attributable to dioxin exposure?
A No.
Q In how many cases would you say the cause is known, the cause of breast cancer is known?
A Very few. They say about 15 percent are related to family history, strong family history. The other 85 percent are of unknown cause, but it is clear

from the epidemiological studies, that it is environmental because when people move from one country to the other, they assume the cancer -- breast cancer risk of the region they move to.

For example, Japanese women have a low rate of breast cancer, but when Japanese women moved to the United States, their breast cancer risk approximates that of a U.S. population. So it is pretty clear that it is related to the environment.

Africa, in the bush, people don't get cancer. They don't get breast cancer. It is unheard of, but we live in an industrial society. We get these cancers.

Q And does breast cancer ever occur in people who have none of the known risk factors?

A 85 percent.

Q 85 percent of the time; that is what you just talked about?

A Yes.

Q Are you aware of something called -- strike that.

Have you ever heard of something called evidence-based medicine?

A Yes.

Q What is evidence-based medicine?

A It's a trick by the insurance industry to not

pay bills.

Q Can you elaborate?

A Yeah. They had a bunch of phony protocols. And if you don't follow the protocol, we don't pay. So it is an attempt by the insurance company to keep your premium and not pay for your medical care.

Q What is the likelihood that an adult female living in the U.S. today would develop cancer today at some point in her lifetime?

THE REPORTER: Cancer or breast cancer?

BY MR. HOPP:

Q Cancer in general.

A The likelihood of getting a cancer is about -- well, if you exclude skin cancer, it is about 30 percent.

Q What is the likelihood that an adult living in the U.S. today would have cancer written on his or her death certificate as either being a primary or secondary cause?

A About 30 -- 30 to 35 percent.

Q Do you agree with the proposition that someone can be exposed to a carcinogen and not get cancer from that carcinogen?

A Yes. We all are exposed to carcinogens constantly. And the body is able to repair the damage

and keep us from developing cancer. So we die of something else, but certain number of people die as a result of cancer as their bodies are overwhelmed, either by being exposed to an overexposure of a carcinogenic agent or susceptibility.

We know that dose matters. The higher the dose, the more likely you are able to contract cancer.

Extensive studies of asbestos workers show a clear dose response. The higher the exposure, the higher the cancer rate. Such that an asbestos exposed cigarette smoker, the risk of getting lung cancer as the cause of death approaches 50 percent.

Q Do you agree with the proposition that someone can be exposed to a carcinogen and develop cancer for reasons totally unrelated to that carcinogen?

A Well, again, we are all exposed to various carcinogenic agents in the environment. So many of those agents don't -- may not be contributing to the cancer that you ultimately develop.

So on a theoretical basis, you might be exposed to a carcinogen that doesn't contribute to your cancer. It is theoretically possible, but we want to talk about details.

As a general statement, you can say it is true, but it needs to be clarified in terms of an individual

case.

Q Are you familiar with aflatoxin?

A Yes.

Q Is aflatoxin a carcinogen?

A Yes, it is considered to be a carcinogen.

Q And it primarily attacks the liver; is that correct?

A Yes. It is thought to be a cause of liver cancer.

Q Can it cause breast cancer?

A Don't know. Never seen any data on that.

Q Are you aware of any recent aflatoxin outtakes in green crops in Mississippi?

A No.

Q Is there any way to model or to otherwise, calculate Sherry Barnes' blood dioxin level?

A No, not that I am aware of. We could -- I have been thinking about maybe doing an extrapolation from the house dust level or soil levels in the homes and see what the correlation with the people living in those homes with their house -- house dust.

Theoretically, you can extrapolate using some technique similar to that.

Q Is the science available to take the facts that we know about Sherry Barnes' body mass index, et cetera,

and the environmental exposure in her home to calculate a blood dose level?

A Yeah. This -- this has been done with lead, for example. Where they take the studies that patients -- they look at their blood leads; they look at the house dust levels for lead; and they then see what the correlation is and construct a model, so that you can predict certain dust levels would result in a blood lead of X amount.

And I have been thinking about doing that with this group, to see what we might be able to say about extrapolation using that technique.

Q Now, in your report, I believe it is -- I'm sorry, Page 49 of 305.

A You want me to look at it?

Q Just read it to yourself. You state that -- you are talking about the 29 people whose blood was taken for the purpose of analysis.

You say subject selected for biomonitoring randomly chosen a total of 103 total residents who were part of the ongoing litigation against the wood treatment plant due to their concern about associated health problems.

And then you say that the inclusion criteria for the randomly selected subjects were 1, above 20

years old; 2, living in the same residence for five years.

Those are the two inclusion criterias you list?

A Yes.

Q Let me go back. How did you come up with the list of 103 residents for the purpose of potential blood level measurements? There are several hundred people involved in this litigation.

A This is in the Columbus case?

Q No. This is in Grenada.

A Grenada?

Q Yes.

A How did the 103 get picked? I am trying to remember. I didn't say. I didn't explain it there?

Q I don't think so. It is Page 49. If you want to look at it.

A These were the 103 that were picked by the attorneys. I didn't participate. I didn't look at a larger group. These were the total number of people that were assigned by the attorneys to be examined.

Q So out of that group of 103 that were presented by the attorneys, you picked 29 based on at least in part on the inclusion criteria that you reference on Page 49?

A Right. After the -- we looked at that, and we

just picked them at random.

Q All right. That is where I am going. I want to make sure I understand the process.

A Yes.

Q Narrate for me then, how did you go from 103 down to 29?

A We asked them -- well, we looked at the questionnaire and we would talk to them and say, look, you are over 20, yes, live with -- what is it, two miles?

Q Same place for five years?

A Same place for five years, and I think within a certain range; one or two miles from the plant. It would have been one mile or two miles.

Q Okay.

A And then we -- I think, Emma Wood, is that the one we talked about yesterday that lived further away than that, but had a real high exposure based on her husband?

Q Husband.

A But everybody else lived within, I think, a certain range from the plant. We tried to make sure that it was, some of the people would be farther away. We just didn't want to just look at all Carver Circle people. We looked at several other people who lived

farther away. But other than that, we did not make any selection.

Q So the three inclusion criterias were age, five years in the same residence, and with the exceptions that you just mentioned, within a certain distance from the plant?

A And we didn't say it was a mile or two?

Q It may be somewhere else in your report.

A I think that is what it was. I think it was a mile.

Q Did you have any other inclusion criteria?

A No.

Q Did you have any exclusion criteria other than not meeting the inclusion criteria?

A No.

Q Well, after you applied those three inclusion criteria, how big was the group? That is, did you get to 29 then applied those three criterias or was there a group of larger than 29?

A No. The people we picked is the people we did the blood on. What we do with the rest of the people --

Q No. Criteria you looked at. And if people met the three criterias, they went into the --

A Okay. We went until 30 people. We ended up at

29. We were limited by how many we could do by the resources available.

Q By the cost? By the budget?

A Yes.

Q Okay. And I am still trying to understand the process.

Did you start with a list of people and go through and see who met the inclusion criterias until you hit 29 or 30, or did you look at everyone, apply the inclusion criterias, and came up with 30 and then --

A 29.

Q -- met them?

A There was more than met them. Once we got our 29 or 30, we stopped.

In other words, there may have been some more people that met the inclusion criteria that we did not test. We did not look at them. Because once we got to the number we wanted, we stopped.

Q So just taking off the surveys off a pile, the surveys' answers --

A As they were coming through the phlebotomist room where the blood, extra blood needed to be taken for these purposes, we screened them --

Q All right.

A -- at the time and we got the people that we

wanted to get.

Q So you got the first 30 who came through the --

A That met the criteria, yes. And by the way, I am looking at the naphthalene data, and it was inhalation and it was respiratory, nasal hyperplasia; but it was also alveolar or bronchial or adenomas or carcinogens. So it was just not nose, but it was also lung.

Q Is this the NPT study in 2000?

A Yes.

Q Is there any other study on rats?

A No. This is on mice about 636 F1, mice.

Q Again, NTP 2000?

A NTP 2000 -- no, this is NTP 1992. This is Table 1. That was mice.

Now, let me see the 2000 paper. Neuroblastomas were also found.

Q In mice or rats?

A That is in rats.

Q And what is the reference?

A NTP, but it is the 2000. Let me see if I can find it. NTP 2000. 49 male and female rats exposed to inhalation, 6.2 hours a day, five days a week for 105 weeks at the rate of zero, 10, 30, or 60 parts per million; and that is when they got not only the lung

cancers and they got a neuroblastoma dose response.

Q At the parts per million range?

A Yes.

Q And so there is two NTP studies that you are relying on for naphthalene than any others?

A That was what -- what California used to derive the slope factor, were these two studies.

Q All right. For the purpose of your opinions in this case, are you relying on any other naphthalene studies that appear to show an increase in risk of cancer?

A Well, let's see. And what can we say about that? The IARC classified that it is a 2B carcinogen in 2002.

Q What is 2B?

A 2B is possibly carcinogenic to humans.

Q And prior to 2002, it was not classified even as a possible human carcinogen; is that right?

A That's right.

MR. PRUDHOMME: And, Tony, for the record there was one exclusion I noted in Dr. Dahlgren's report on Page 49, and that was none of the members worked at the wood treatment facility.

MR. HOPP: That was the exclusion?

MR. PRUDHOMME: That was the exclusion factor.

MR. HOPP: Thank you.

THE WITNESS: Other factors that would indicate --

BY MR. HOPP:

Q Well, other studies?

A Other studies that would support that is carcinogenic.

Q I am aware of a couple of animal studies. I want to know if you have any animal or human studies that support that naphthalene is either an animal or human carcinogen?

A No. Let me look at this.

In the Crisp, C-R-I-S-P, study, this scientific database is maintained by the public health service and they list various studies. I don't know. Maybe I should look through this later.

Q Okay. Maybe that is something that we can come back to. Just to finish on the topic of naphthalene, old style moth balls were made of naphthalene; correct?

A They were. And they were banned because of the concerns about its cancer-causing capacity.

Q How long ago were they banned?

A In California? They were banned -- all pesticide registration of naphthalene including moth repellant was canceled in 1991.

Q I know that I bought naphthalene moth balls in Naperville, Illinois after 2000 because I have them in my garage.

A Well, you could not buy them in California.

Q But you could buy them in other places even now, if you know?

A You just told me that you bought some. So I suppose Illinois did not ban them, I guess.

Q But the moth balls that everybody's grandmother used to use, those were naphthalene; right?

A Yes, that's right.

MR. HOPP: Shall we break for lunch?

MR. PRUDHOMME: That's fine.

(Lunch recess.)

BY MR. HOPP:

Q Dr. Dahlgren, referring your attention back to page 49 of 305 of your report, this is where we were looking at the notion of choosing the test subjects.

A Yes.

Q You state that the subjects selected -- let me just read it. "The subject selected for

Biomonitoring were randomly chosen

From a total of 103 residents, were

Part of an ongoing litigation against

the wood treatment plan due to their

concern of associated health problems."

So the 103 people who came through the testing center you described before lunch were already plaintiffs or potential plaintiffs in litigation; is that right?

A Yes.

Q And were they all ill or were some of them ill and some of them were concerned about being ill?

A Both. Some were ill. Some were concerned about being effected in the future.

Q And -- strike that.

Did each of these 103 people fill out your questionnaire?

A Yes.

Q Do you have a list somewhere of the 103 people from whom you selected the 29?

A Yes, I'm sure I do. I'm not sure if I have it with me today, but I think I do have a list.

Q I will follow up with a letter to Keith, but I will make a request for the list of the 103 people from whom the 29 were selected.

Where was the blood drawn done for the 29 people from Grenada?

A We rented a hotel. I am trying to remember what hotel it was.

Q It was in Grenada somewhere?
A In Grenada.
Q So these people were not bussed to Miami?
A No, they weren't.
Q Okay. And were there specific blood collection procedures that you had to observe for the purpose of dioxin testing?
A Yes. ERGO sends us glassware and instructions of how to handle the blood.
Q Was there a local phlebotomist you used who then collected the blood and followed ERGO instructions?
A No. It was a phlebotomist who I brought with me; actually, two women who, I believe, in Grenada. They were the people from Lake Charles that we used in phlebotomy for years now.
Q What are their qualifications?
A They are professional phlebotomists.
Q Do you know their names?
A Betty and -- what is the other lady's name? I don't remember.
Q And these are technicians from Lake Charles, Louisiana?
A Yes. Correct, that draws the blood for us when we do study in the fields.
Q I take it, that it is important to follow

ERGO's instruction for collecting the blood and preserving it for shipment?

A Yes, it is quite an elaborate procedure because we ended up sending the blood on dry ice.

Q Do you send whole blood on dry ice or do you spin it down to serum before you send it?

A Spin it down and separate it and put it on the dry ice and then ship it in a special glassware.

Q Was there a lab, then, that these phlebotomist used for these purposes?

A We have our own centrifuge. That is all we need.

Q So you actually brought the centrifuge with you and set it up at the examination site?

A Yes.

Q What -- strike that.

If the samples are improperly preserved, if one of the technicians, for some reason, makes a mistake, how could that impact the results of the sampling?

A Well, you could, I suppose -- I am trying to think what kind of a mistake we would talk about.

Q Well, let's just say, for example, the samples warm up and they are not frozen or they are not cold enough by the time it reached West Germany -- I guess, now Germany?

A Yeah, they don't distinguish west and east any longer.

Q That's right. I am showing my age.

A I always thought that the dioxins are exceedingly stable and as we were talking yesterday, you can keep them in a freezer for years and still get reliable results.

I don't know what the effect -- the reason why you don't want to get it warm is you can get bacterial growth and bacteria might -- might metabolize the dioxins a little bit. That is why you keep them frozen because you don't want any microbial action to reduce your, you know, the anolytes of interest.

So, I guess, that is the point I would make is that if they got unduly defrosted, there might be some errors introduced, which would tend to reduce the values.

Q Let's talk about the PAH and DNA adduct study.

Are there geographical variations in the blood level of PAH, DNA adducts in the United States?

A Yes.

Q Can you describe what those variations are?

A Yes. The biggest difference is urban versus rural. If you live in an urban area, you tend to have higher adduct levels than if you live in a rural area.

We talked about that yesterday.

If you live close to a roadway, you are more likely to have elevated values than if you lived further away from the roadway. And I think those are the major regional or geographic differences that have been described.

Q Is there any sort of general distinction between DNA adduct levels -- background DNA adduct levels in Mississippi as opposed to Florida?

A You wouldn't expect that if they were in similar size towns, as we discussed yesterday, as well.

Now, there may be a difference -- the urban, rural differences are not great. There are some slight differences. There may be -- let me just look at this paper.

I think I see where it went. It is right here. There is a review paper on this urban, rural difference.

Q Is that one of the papers you cited in your recent bibliography?

A Yes. Let's see which one was it. I guess, it's the Kriek '98 might be the one that I am looking for.

Here is my list. Okay. Relevant -- this is Kriek, K-R-I-E-K, 1998. And he has got a review of a lot of the different studies. I thought he had an

urban, rural distinction in one of his tables, but I am not finding it right quick.

Here we go. Well, interesting study. He doesn't quite do what we want because there is a -- bus drivers looked at in --

Q Bus drivers what?

A They looked at bus drivers.

Q They have higher exposure?

A They have very high exposures from bus driving, and the one here with environmental exposures, they are mainly talking about summer and winter differences.

Q And that is a relevant distinction, people tend to have higher DNA adduct levels in the winter; is that right?

A Yes.

Q Is it because they are in the house?

A Yes. And there is more -- in this study, anyway, there is more burning of fossil fuels to keep warm. This is in Poland. The difference between summer and winter is approximately a doubling of the level in the exposed population; but there is no difference in the control group between the winter and summer.

Q Okay. Eric Kriek, is that the name?

A K-R-I-E-K, and --

Q Mutation Research 1998?

A Mutation Research '98, yes, that is the paper.
Q What table are you on for your --
A We are looking at Table 3. And let me see, there are some other papers that address this, too.
Q This Table 3 looks both at P32 post-labeling and Alyssa techniques. That's correct.
A 2000 Perera.
Q If you look at the Perera for the urban, rural distinction?
A Well, I think she did show -- she discusses it in some of her papers. Let me see if I can find the one quickly about this issue.
Q This is Frederica Perera?
A That's right. She has probably written more on this subject than anybody else. P-E-R-E-R-A. She discussed breast cancer in PAHs in this paper.
Q Which paper?
A This is 2000, Perera 2000. I am just looking for her discussion of our point, but environmental susceptibility versus exposure, which we were discussing, she addressed that issue, also.
This is just an old point. Maybe I will go back to the older papers. And there is a significant difference in the Hemicky paper, 1990, talked about urban, rural differences.

Q Kari Hemicky?
A Um-hmm. I think I have that paper on another file. I am not finding it.
Q But, generally speaking, you think there is a slight distinction between urban and rural residents in effect to PAH, DNA adducts?
A Yes, there is a difference.
Q Exposure to various sources of PAHs is going to effect the level of someone's PAH, DNA adducts; is that right?
A Yes.
Q And that is why cigarette smoking increases the level of PAH, DNA adducts in someone's blood?
A Correct.
Q Also, exposure to side stream smoke?
A Yes.
Q Secondhand smoke?
A Side stream/secondhand smoke will increase the risk.
Q And if someone does household burning of waste or leaves, that would also increase their risk -- or I am sorry, their level?
A Their level of PAH adducts, yes, can be increased by burning of carbonaceous materials.
Q Now, do you know how the daily dose of PAHs

from cigarettes smoke compared to daily PAH dose incurred by one of the plaintiffs in this case from creosote smoking?

In other words, the --

A The smoking effect?

Q Yeah. What would the smoking effect be?

A It is very, very slight. Even in this case, you can see, if you look at the paper, we have a few current smokers and they were not any different than the other smokers and -- I mean, nonsmokers. That is what all of the studies have shown. A very slight difference.

It is not as important as the urban, rural difference. However, if you want to look at smoking, and if you look at the papers that have been published, they may indicate that there is a slightly, higher level in smokers. Not all of the studies have shown that, but some have.

Q Are you familiar with an experimental concept called a positive control?

A Yes.

Q Would you consider cigarette smoking a positive control for detecting PAH, DNA adducts?

A Let's look at our sheet. Which exhibit was it that had the DNA adducts? Because it really wouldn't

work as a positive control.

Q That's 68. It does indicate smoker and nonsmoker. Here is my copy.

A See, if you look at current smoker levels, clearly, you know, you got Gloria Loggins. She is 2.74. Glenn Collins, 5.44, which is the highest value -- no, Randy Barnes is the highest value.

Q And he is a nonsmoker?

A He is a nonsmoker. Sherrie Ratliff is a current smoker and she is only 2. So if you look at those, it does not look like smoking has any impact.

Q Is that consistent with what the literature indicates?

A Yes. Yes, as I said, most of the studies have concluded that smoking is, you know, not the main source.

Q Do you know the average daily exposure of PAHs of a nonsmoker?

A The average PAH level?

Q Yeah, in nonsmokers?

A It is not -- we don't have the numbers like we can talk about dioxin TEQs. We don't have that same luxury here because, as I said, there is variability in the way it is done. So that there is no defined value out there for normal and abnormal.

Q No defined background level?

A No defined background level in terms of the number type thing. There is a general range, but, you know, how many new -- how many adducts per 10 to be nucleotides.

Q Do you know a range of variation in PAH, DNA adducts in an individual day-to-day -- bad question. Let me ask it again.

Do individuals, you or me, for example, have --

A Day-to-day variation?

Q -- day-to-day variation In PAH, DNA adduct level?

A No -- well, what we do know is that it is attached to the lymphocytes and that is what we try and look at among the nuclear cells. And that includes monocytes and lymphocytes and they tend -- monocytes tend to have a fairly short half-life, but the lymphocytes have a long half-life.

The bulk of stuff you look at is, you know, 25 to 40 percent of the cells are lymphocytes and those have a long half-life. So they are not likely to change radically from day-to-day unless there was a big spike of exposure.

In the studies of smokers who stopped smoking, they can have quite high levels and they follow them

through to see how long it took the adducts to go away. I was just looking at that. It takes about two months for them to go down.

Q You said lymphocytes and --

A Monocytes.

Q Those are white blood cells; correct?

A Yes.

Q And when you do these PAH, DNA adducts studies you are actually looking for PAH, DNA adducts in white blood cells; right?

A Yes.

Q You are not looking for them in liver cells and breast cells?

A No. The blood is the easiest tissue to get. I mean, obviously, there have been studies on these other tissues, but the ones that we are talking about here that we did in this case were done on white blood cells.

Q And going back to your earlier answer, you said that the two different types of white blood cells have different half-lives. What are those half-lives?

What is the half-life for lymphocytes?

A Well, the lymphocytes half-life varies. There is a small segment of long lived lymphocytes who actually are in the blood stream for two to three years.

They are memory cells. And then there are

lymphocytes that have a half-life of about two to three months, and that is the bulk of it.

Q How about the other type of white blood cells who you said have a shorter half-life?

A The leukocytes, those are the polymorphonuclear leukocytes. They have a shorter half-life, in a matter of hours.

Q Now, the P32 post-labeling technique, how specific is that technique for PAH adducts?

A It is very specific for PAH adducts. In other words, you are asking would it cross-react with adducts formed by other chemicals like, let's say, atrazine.

Q More specifically, can it defect other bulky DNA atoms?

A My understanding is that the bulky adducts that are detected by this method are PAH and I am not familiar with what might be giving additional signals that are not PAHs.

I don't know how pure, how specific the technique is. It is my understanding that it is very specific, but the percentage of specificity, I don't know.

Q All right. You state, on Page 47 of your report, that PAH leave characteristics, fingerprints when they bind to mononucleotizing DNA.

Were you able to identify any PAH fingerprints in this case without being able to determine patterns of PAH, DNA adducts, and how they vary between these exposed and control groups?

A You would have to talk to Dr. Phillips about that. He is the author of the opinion that these things are specific.

Q Okay.

A And we don't know which PAHs they are, but we know that there are -- you know, I think mostly like 90 plus percent PAH adducts and not adducts of other types.

Q On Page 50 of your report, you state you did not adjust for dietary confounders. And then you say,

"Barbecue intake, because
that history was unavailable
at the time in our comparison
group."

A That's right.

Q What would be the magnitude of PAH, DNA adduct levels you would expect in a regular consumer barbecue?

A I don't know. Because I looked at these papers, I was looking for someone to try to quantify barbecue. And I know there is -- I read a paper on it at one point in the distant past, but I could not put my hand on it recently.

Q Are you aware of any peer-reviewed published papers which demonstrate an association between creosote PAH, DNA adducts in white blood cells and human cancer?

A Where the source of the PAH was creosote?

Q Yes.

A No.

Q How about generally, are you aware of any peer-reviewed papers that show an increase in PAH, DNA adducts in white blood cells and human cancer?

A Yes, there are a number of studies that have shown that.

Q And are those in your bibliography?

A They are in the bibliography. Perera, the one that we just looked at, has a whole section of her paper on the association of DNA white blood cell adducts and human lung cancer.

Q Lung cancer?

A Human lung cancer and human breast cancer, both.

Q Which Perera paper was that? What year?

A I think I was looking at it a second ago. It was '99; wasn't it? 2000.

Q Well, you got it up. What is the title of that Perera paper, 2000 paper?

A Molecular Epidemiology, On the Path to

Prevention.

Q Are you aware of any peer-reviewed public study that demonstrates an association between environmental creosote exposure and increased PAH, DNA adduct levels in human white blood cells?

A No, I don't think that anybody has done this using -- where creosote was the source of exposure. Coke oven workers have been studied. Smokers have been studied. People living in Silesia, Poland has been studied and a whole host of other people studied using white blood cells; but I don't remember any of them having creosote as the source.

Our paper, when we finally get it published, will be the first peer-reviewed article where PAH adducts have been measured in a creosote exposed population.

Q And are you currently writing the paper?

A We are working on the expansion on the paper that we talked about yesterday.

Q Biomonitoring paper?

A Biomonitoring paper, yes.

Q Who are the authors going to be on that one?

A Well, myself, Dr. Schmidt, Dr. Anderson, Harpeet Tarkar, and possibly Dr. Philips. And I'm not sure who else might be added to the author list.

Dr. Sposs from my office may be added.

Q Are you aware of any peer-reviewed published studies that demonstrate that living on PAH contaminated soils can increase PAH, DNA adduct levels in white blood cells in human?

A That is something that we are going to look at to see if there does seem to be any trend from the PAH adduct levels we found in the house dust and in the soils of these various homes to see if there is any linkage to the PAH adduct levels that we found.

Q What effect do polymorphisms in xenobiotic metabolizing and detoxifying genes have on white blood cells, PAH, DNA adduct levels in humans?

A There is an effect. Again, we can go to that Kriek paper. In the Kriek paper, there is a Table 4 looks at different polymorphisms and there appears to be a difference.

For example, in those individuals who have an enzyme that is CYP1A1 BAL positive/negative, those ten patients had adducts that were significantly higher than other types, other polymorphisms.

And then if you look down to coke oven workers, the ones with the very highest adducts were ones that had a CYP1A12A/2A-GSTM1 null. That GSTM null 00 indicates that they were deficient in glutathione

metabolizing enzyme and that caused their adducts to be very high.

They were 44, where as some of the other coke oven workers were -- but there was only one worker who had that polymorphism. So we do not want to generalize too much from it, but it was strikingly high.

What it means is that that person with that defect was not able to process effectively the adducts and get rid of them and repair the DNA. So the DNA adducts built up to a higher level in that particular polymorphism.

Q So depending upon your genetic makeup, you could have a particular sensitivity to PAHs?

A Yes.

Q Do you know what types of polymorphisms the plaintiffs in case had or has?

A No, there is no data on what their various genetic patterns are.

Q How great an increase in cancer risk do you believe is associated with an increase in PAH, DNA adducts from 0.75 per 10 to the 8th nucleotides to 4.11 per 10 to the 8th nucleotides in white blood cells?

A You mean how much difference in risk would there be indicated by those two levels?

Q Right. If you go from .75 to 4.11, what is the

jump in the risk level or is that something that has even been calculated?

A I have not seen anybody calculate it using that technique. What they usually do is they talk about the population of people who have higher values as opposed to a population of people of lower values and the risks in the two populations.

I don't -- I have not seen anybody really zero in on an individual patient and say, okay, their value is three and their value is seven; and, therefore, that person has got two-and-a-third times higher risk of getting cancer. It isn't that precise.

Q Okay. And you have not seen anybody generalize on risk levels for human cancer based on PAH, DNA adduct levels? Apart from, you said the single patient in your prior answer.

Has anybody published a slope --

A That was a single patient who had the higher adduct levels after being exposed to the coke ovens and had a particular polymorphism.

The point that there are -- I mean, every study practically in here reports a higher rate of cancer in the people who have higher adduct levels.

Q Sure. Is there a slope factor that you know of that is accepted for PAH, DNA adducts and cancer risks?

A No. As I said, I don't think anybody has worked that out. What they have looked at is groups.

Q Now, PAH, DNA adduct levels that were detected in your study were in circulating white blood cells; correct?

A Yes.

Q And circulating white blood cells cannot develop in the cancerous cells because they are terminally differentiated; is that right?

A They are terminally differentiated cells. Therefore, they cannot become cancer.

Q Right. They can't -- well, let me ask you generally. Do white blood cells in circulation become cancerous?

A No, the -- no, I don't think so. I mean, the leukemias come from earlier cell types. Obviously, then, circulating a cancer cell in a leukemia patient, but if you have a normally developed cell, it is not going to undergo cancers degeneration from what I understand, anyway.

Q Well, in part, because -- correct me if I am wrong -- white blood cells, once they are in the bloodstream, don't multiply?

A Well, I am trying to remember. There are some changes that they can go through, but I think,

generally, you are right.

Q And in the 29 people in Grenada, you did not measure PAH, DNA adducts in other tissue; is that correct?

A Correct.

Q Now, on the second table of your report that is Pages 52 through 53, you show the results for 24 people who underwent PAH, DNA adduct testing.

And then you state that 5 of the 29 randomly selected plaintiffs failed to show up to have their blood drawn.

In your experience, is a 17 percent refusal rate unusual?

A Usually it ranges between 10 to 15 percent. So it isn't too far out.

Q Do you believe that the 17 percent refusal rate in this case affected your results at all?

A I don't think so. I mean, it is kind of hard to know why they didn't want to do it, but --

Q But on Table 3 of your report, you present demographic data for all 29 as opposed to just the 25 that showed up; right?

A Right.

Q Can you tell me which people didn't show up?

A Well, we can look at those two tables and

figure that out. We have the dioxin table and we have the PAH labels to see what is missing.

Q If you compare birthdays, you can figure out who they are?

A Yeah.

Q We will save that exercise rather than take the time.

So does Table 3 represent the -- I guess, I am confused.

You got demographics for 29 people in Table 2 and then you got Table 3. Does the -- do the averages or the mean levels that you calculated reflect just the measurements in the 25 or is it all 29?

A For the adducts?

Q On Table 3. Does that relate to just the people who were measured or does that relate to everybody?

A Well, it looks like it relates to -- something is a little off here. It should be 24 people. It must be just a mistake in the table. We have to fix that because it looks -- refers to 28 and one missing race.

So it refers to 29, but the adducts were only done in 24. So that doesn't make sense. This is the demographics of the whole 29 and not of the 24 that were tested for adducts.

Q And do you think the mean adduct level would go up or down if you subtracted the four that didn't show up?

A Well, what value would you assign them? You wouldn't -- you would not assign them a value because you wouldn't have any idea where they stood. But I mean, you assign them the mean value, it would not change anything.

Q On Page 47 of your report, this is the next and last sentence of the page, you state, "PAH,

DNA adduct levels in white blood

Cells reflect environmental exposure

To PAHs," and then you cite Haugen for that and Phillips.

A Okay. What page?

Q Page 47, it lists Footnote 77 and 78.

A PAH adduct levels and reflects environmental exposure, okay.

Q And the references are Haugen, H-A-U-G-E-N, and Phillips. Was it the Haugen paper a coke oven workers study?

A I will have to look and see. I don't remember from memory. Should we look at Haugen?

Q Yes, if you could confirm it to me. You can probably look at your footnotes in your paper.

A Is there footnotes? Where are the reference pages? I forgot. It is back there somewhere. I think it might be faster.

Q H-A-U-G-E-N, 1986.

A Right. Frustrating.

Q It is not cited in your bibliography; Haugen?

A Where is it? It should be here under coal tar. I don't see it. Well, I got to find the reference.

Q Let's move on. The Phillips paper, which you also cited to support that point, is Phillips 1990; is that right? While you are looking at the references.

A Phillips 1990 is right here.

Q And the Phillips 1990 paper examined 31 heavy smokers and 20 nonsmokers; is that right?

A Let's see 37 smokers, eight former smokers, and eight nonsmokers; is that the right paper?

Q Right. 31 of the people he looked at were in excess of 20 cigarettes a day?

A Correct.

Q I want to turn now to the paper cited in your report, specifically in reference to breast cancer.

If you remember your report contained a main section and then a patient reference list?

A Yeah.

Q And there is a reference for each patient?

A Yes.
Q Sherrie Barnes, you have a list of breast cancer references -- and correct me if I am wrong -- it appears to me, at least, that the breast cancer references for Kay Hobbs, for example, are the same for the references for Sherrie Barnes?
A Well, that would make sense.
Q So it is the same papers. The first one you cited was Brown 1998; correct?
A Um-hmm.
Q And we already looked at that. That is deposition Exhibit No. 130?
A That's correct.
Q One, the next one is Corinne Charlier, C-H-A-R-L-I-E-R. We are at 131; right?
(Defendants' Exhibits 131 was marked for identification by the court reporter.)
MR. PRUDHOMME: You are at 131 -- the next one would be 131.
BY MR. HOPP:
Q I am handing you a copy of the Charlier paper that we have marked as 131. Is this the same paper that you have cited?
A Yes.
Q And this deals with PCB contamination in women

with breast cancer; is that correct?

A Yes.

Q And what did Charlier conclude? What I have given you, I think, is an incomplete copy.

A Relationship between PCB concentrations in serum and risk factor was mainly due to serum levels PCB 153, which was significantly higher in breast cancer women than in diseased-free subjects.

1.63 versus 0.63, even after accounting for other potential risk factors, these results suggest environmental exposure to PCBs may contribute to multifactorial pathogenesis of breast cancer.

Q Now, in the group that Charlier studied, I am looking at Page 179.

A Um-hmm.

Q The prevalence of menopause was significantly higher in the woman with breast cancer; is that right?

A Yes.

Q Also -- and this is further down the page.

Also, for PCBs 52, 101, and 180 serum concentrations did not differ between the two groups; is that right?

A Help me out here. Where are you?

Q This is under PCB Concentrations, Page 179.

A Okay. Yeah, I read that in the abstract the

153 and 138 were higher in cases in control and total PCB content was also higher.

Q In cases?

A Yes.

Q Okay. Looking, again, at 179 under the heading Association with Breast Cancer, she states.

"High concentrations of PCB 153 were significantly associated With an increased risk of breast Cancer despite the presence of other factors"; is that right?

A Um-hmm. Right.

Q So it was the presence of that single PCB that she identified as the risk factor for breast cancer; is that correct?

A Um-hmm. Yes. That's right.

Q Looking at the conclusions, I am on Page 180, it is toward the end above Table 3, it says,

"In conclusion, our results Comfort the debate that there Is not sufficient evidence to Answer the question on human Risk resulting from low-dose endocrine-related effects."

Is that a typo or do you know what that means,

"comfort debate"?

A I have never seen that phraseology. I am not sure what he meant. Results comfort -- I don't know. I don't know.

This is a Belgium who is not a native speaker of English. He may have thought of something he was trying to say.

Q And then what Charlier recommends is

"Further interdisciplinary research, combining detection and quantification of pollutants, epidemiological data collection, but also metabolic polymorphism investigations"; Is that right?

A Yes.

Q Does the Charlier article include relative risk data for breast cancer?

A Well, it has the odds ratio here. Multiple Logistic Regression Table 3. Basically, PCB 153 is elevated, your odds ratio is 1.8 and it is statistically significant.

Q To what extent is PCB 153 dioxin-like?

A I don't remember what its TEF is. Let's see if we can figure that out. I may have put it on my -- I probably didn't put it on my table to make it easy. So

we have to look somewhere for it. I'm pretty sure I didn't put that one in my -- no, I didn't include it. So I have to look it up.

Looking for the table with the TEFs in it. And, hopefully, we will find it. I can't find it.

Q All right. I don't think we are going to finish today. We are going to have to return on that subject.

But in answer to the question to what extent PCB 153 is dioxin-like the answer, by its TEF; is that right?

A Yes.

Q Next one is Demers, D-E-M-E-R-S, 2002?

A Okay.

Q I am handing you what we have marked as Exhibit 132.

(Defendants' Exhibit 132 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is a copy of the Demers article entitled Plasma Concentrations of Polychlorinated Biphenyls and the Risk of Breast Cancer: A Congener-Specific Analysis.

What did Demers conclude?

A Cases had significantly higher concentrations

of PCB 99, 118, and 156. Associations were found between breast cancer risk and PCB 118 or PCB 156.

Breast cancer risk was also associated with total concentration of three monoorthosubstituted congeners. 105, 118, and 156, TCDD paradioxin toxic equivalence with the highest concentration of 2.02, fourth vs. first quartile.

These results suggest that dioxin-like PCB increases breast cancer risk. Alternatively, the results may be explained by differences between cases and controls regarding metabolic pathways involved in the transformation of both monoortho PCBs and estrogens.

Q What does that mean, the alternatively?

A It is the susceptibility issue that they can't handle PCBs as effectively. You know, therefore, they have higher concentrations because they cannot excrete them efficiently.

Therefore, they go on to have the adverse effect. As opposed to patients who can get rid of them more effectively.

Q And Demers concludes, this is at the very end of the paper, "Although levels of these

Dioxin-like compounds may

Present a risk factor for the

Disease, additional studies are

Needed before concluding that
These compounds are causally
Involved in the etiology of breast
cancer"; correct?

A Yes. That is what all academics always say, we need more studies. Standard procedure in almost every paper.

Q Fair enough. But Demers is not willing to commit to the definite conclusion that they have demonstrated a risk between these exposures and these diseases; correct?

A That is what he says, yes.

Q And is this a case control study?

A Let's see, they identified 315 women for breast cancer and then recruited 219 controls at four different hospitals for the first control.

The second control was 307 women selected randomly from the general population of Quebec. Case controls were then matched for age into five-year age groups. And region, rural versus urban.

Cases were excluded that they showed distant metastasis of diagnosis or if they had a previous history of breast cancer or other cancers, et cetera.

Q So they attempted to match cases with controls?

A Yes, they did, although there was a little bit

of a cross-sectional aspect of it, as well.

Let's see, what did they end up with? How many controls did they end up with at the end of their process?

Selected characteristics on Table 1. 314 cases at 523 controls. So it looks like they just added them together, at least for the demographic study.

Yeah, it doesn't look like they excluded anybody from their control group, but they did -- as part of their analysis, they looked at different age groups and compared groups and age, 30 to 35.

In cases and controls for the various use in -- I don't see where they talk too much about age after that. They are mainly talking about the PCB levels after that.

So, anyway, it's a very large case control study where they had almost twice as many controls as exposed. And, you know, I think it is sort a combination cross-sectional and case control study.

Q On Page 2 of the study, Page 2 of 13, Demers states -- he talks about previous studies, since the early 1990's. It says, "Most studies that

used the sum of all PCB congeners

as the measure of exposure did

not report an association with the

risk of breast cancer."

Do you agree with that statement?

A Well, I think the statement is correct. He has got one to seven here. These are the earlier studies where they use total PCBs.

Q Right. So if you --

A That is using the Webb-McCall technique. It only quantifies a fraction of the PCBs anyway. So it is really a lousy way of estimating PCB fiber.

And what they have done here and other studies that are broken out in other specific congeners, that is where they started to see the effects.

Q Right. And it actually goes on to say that,

"However, a series of recent studies that examined the relationships with individual PCB congeners or Groups of congeners have yielded conflicting results."

Do you agree with that statement?

A Well, we have to look at each paper, but the statement, obviously, is what he said. Whether I agree with it or not, I guess we would have to go through each paper to see.

I think there are some negative studies. I just don't -- you know, that is usually the case. There

is usually positive and negative studies, and I think that is all he is saying.

Q Again, looking a little further down, this is on Page 2, five lines up from the bottom of the page, it says, "On the one hand, the dioxin-like

Compounds elicit a broad spectrum

Of antiestrogenic activities and may

reduce breast cancer risk."

Do you agree with that statement?

A Yes, we talked about that this morning. And, again, it gets back to this question that Dr. Burnbaum brought up, which is that what we really want to look at is the time of exposure and maybe that is one of the confusing things.

If we look at a patient with breast cancer already, we may not be looking at the right time.

Q Now, Demers did look at serum levels for individual PCB congeners for the cases and controls; correct, that is Table 2?

A Yes, and he selected the PCBs. I think that's probably more numerous and have the dioxin-like toxicity and the so-called monoortho and coplanars.

Q And at what exposure level, if any, did Dr. Demers identify an increase risk of breast cancer for the congeners that he associates with the increased

risk of breast cancer?

A You mean what was the level of the PCB?

Q Yes. How much was enough to increase your risk above the odds ratio above one?

A Let's see. The differences between cases and controls, I guess, there is a difference PCB 99 -- it is the ones he identified, 99, 118, 156. He didn't find 153 elevating the risk.

Q In contrast to Charlier or who did?

A Yes. But I think I could tell you, it is possible that they misidentified them. Anyway --

Q You think they may have misidentified a congener?

A Well, it is possible. You know, it's a -- wait, we will see.

Further studies, I'm sure are going to be done. I haven't gone through and looked at the -- all of the studies in detail asking that question about 153 versus 156.

Q The question, though, pending is did Demers identity a level of any particular PCB congener in the blood which would necessarily result as an increased risk?

A Well, I'm not sure that he exactly -- yeah, he just says as the TEQs go up, the risk goes up. I don't

see that he quantifies that risk in terms of saying what level of PCB you need.

And, again, we go back to the Birnbaum argument. It is probably not the level of PCB level that she has today that is the culprit. It is probably PCB exposure over time; but what is important is that there is this consistent finding of PCBs and breast cancer in study after study after study. And where there is smoke, there is probably a fire.

Q Next one you have cited is -- I don't know how to pronounce it, I guess Dusich, D-U-S-I-C-H. Dusich?

A Yes.

Q This is 133.

(Defendants' Exhibits 133 was marked for identification by the court reporter.)

BY MR. HOPP:

Q I am handing you what we have marked as deposition Exhibit 133, the Dusich paper entitled Minnesota Department of Public Health, Cancer Rates in a Community Exposed to Low Levels of Creosote Components in Municipal Water.

Can you tell me generally what Dusich concluded?

A Well, what he concluded is that there was an increased rate of breast cancer associated with the

contamination.

Q This is the study of St. Louis Park, Minnesota?

A Yes.

Q And somewhere near St. Louis Park, Minnesota there was an old wood treatment plant; right?

A Yes.

Q And there were PAHs in the ground water in St. Louis Park?

A That's right.

Q But the PAH concentrations were detected some time in the 1970's or '80's and no one knows how many years that contamination was there; correct?

A Correct.

Q And Dusich states there -- this is on page -- the first page of the article near the bottom of the first column, "There appear to be no

Epidemiological studies of human populations exposed to low Levels of PAH in water supplies."

Do you see that?

A Yes.

Q In fact, Dusich is probably one of the only studies, if not the only study that examines that; right?

A I didn't find any other others, no.

Q Except the Dean paper which followed; right?
A Yes, that's true, also. The Dean paper did not address this same population.
Q Now, Dusich found an increased incidence of breast cancer, and I think a weak association with gastrointestinal cancers; is that right?
A Yes.
Q But she found no other increases in cancer rates; is that right?
A That's right.
Q So it is negative for every cancer other than breast and GI?
A That's correct.
Q Did Dusich ever calculate the relative risk for breast cancer?
A Isn't it right here somewhere? Let me see. I think it is. Let's see. They have a 1.5 full difference in rates. So I presume he -- he is not terribly clear the way he writes it, but it appears the relative risk is 1.5.
Q But it is not expressed as a relative risk calculation; correct?
A Well, he expresses everything else as a relative risk.
Q Right.

A Anyway, he states here -- you got almost backhandedly, he says, because of the sizeable population of Jewish ancestry estimated to be 20 percent in 1971, the influence of this factor as a particular interest, but would not explain the 1.5 fold difference in race even if 20 percent of St. Louis Park's breast cancer cases were Jewish, and a twofold relative risk existed.

So by implication, there was 1.5 fold increase.

Q Okay. But -- and I have to confess. I have a little trouble interpreting that sentence and I think maybe you may have expressed the same concern.

A Yes. Well, here is relative risk down here. It is at the bottom of the table, it says, Comparison, St. Louis Park versus Edena, breast cancer, 3.38.

Q Okay.

A P value, 0005.

Next, St. Louis versus Richfield, 10.85, .001. St. Louis Park versus SMSA, 13.64, so those are very high relative risks.

Q So that column, 3.38 and 10.85 and 13.65, those are relative risk numbers?

A Yes. Comparisons with different population. You see up above it says St. Louis Park, Edena, Richfield and MSP SMSA. I think that is Minnesota state

rates.

Q It is the standard Metropolitan statistical area for Minneapolis, St. Paul.

A I see. Compared to those three other groups, you get different relative risks depending on which group you are looking at.

Q What is the relevance of the P value?

A That is the degree of statistical significance. Anything that is greater than .05 is considered highly significant.

Q And so the only P value that is higher than .05, according to Dusich, is St. Louis Park versus Edena; right?

A Yeah, he has listed this as .05 -- less than .05. P less than .1. I think what he meant to say was it was between .05 and .1.

I think he made a mistake or she made a mistake when she expressed that table. But I think that is what she is meaning there.

Q But the St. Louis Park versus Richfield and St. Louis Park versus SMSA, those are not statistically significant; correct?

A No, no, no. Those are highly statistically significant. .05 or less is considered highly statistically significant. So all the rest of those are

highly significant statistically. At a very, very high level of certainty, that is statistically significant. Borderline.

Q In our case; that is, in the Grenada case, the exposures were not due to ground water; correct?

A As far as we know. Now, there were some personal wells that people drew water from, but they were never measured.

And, apparently, they -- most of the people were still on municipal water. They apparently did use some water from a local well from playing in it and so on, which was eventually closed; but we just don't have any data.

Q And in particular with respect to Sherrie Barnes, you don't know whether she was ever on well water; right?

A Correct.

Q Does the Dusich study isolate the level of creosote in ground water which is necessary to cause an increase risk of breast cancer?

A No. All they said in this paper is that there was levels considered to be above the MCL.

Q And what is the MCL for PAHs in ground water?

A Don't know offhand.

Q Just, for the record, what is an MCL?

A Maximum contaminant limit.
Q And that is the limit that is set by the United States EPA; is that right?
A Yes, and sometimes by state or local governments.
Q And the idea there is it is an acceptable level of a particular constituent in ground water; is that right?
A Yes. Again, you go back to this whole issue of regulatory values, which are set and it doesn't mean they are necessarily safe, and there would be no adverse effect below that level because their knowledge is constantly evolving, A, and, B, sometimes they set those based on economic issues.
Q All right. Let me hand you 134, which is the Dean paper.
(Defendants' Exhibits 134 was marked for identification by the court reporter.)
THE WITNESS: Yes. I didn't include this in my bibliography because this paper is a joke.
BY MR. HOPP:
Q All right. Let's talk about that.
A What they did is they eliminated people who were complaining of environmental worry, and when they excluded them from the cohort, which they did, they

found no significant difference. No one that I ever heard of would ever do anything like that. It is just ridiculous.

Q All right. Dean Dusich?

A Dusich isn't on this paper.

Q Yes, she is. She is the third author on the Dean paper. So --

A You're right.

Q So looking at deposition Exhibits 133 and 134, the Dean paper is 134 and the Dusich paper is 134; they have authors in common?

A No, no. It is the same cohort.

Q It is the same cohort and same authors?

A Same cohort -- well, two of the same authors. But what difference does that make? The point is this is the same cohort. They just reanalyzed their data. You see, they got Harriet Imrey instead of Eunice Sigurdson.

Q Right.

A It is on both of them.

Q Kari Dusich, William Hall, and Andrew Dean are on both papers?

A Yes.

Q And the Hall paper retracts the finding from the Dusich paper; is that right?

A Yes, using the trick that I just told you they use. That is ridiculous. I don't know how they ever got this thing published. It is ridiculous to eliminate people because of environmental worries is nuts.

Q Is that the only reason they eliminated people?

A Um-hmm.

Q Didn't they actually look at a larger control group in the Hall paper?

A Yes, but that would not eliminate the problem that I am referring to.

Q So you reject the Hall paper out of hand?

A Out of hand. Absolute garbage. This is unbelievable.

Q Even though it is authored by the same people who authored the Dean paper?

A I know what happened here.

Q What happened?

A They got pressure from their bosses to reanalyze the data and get rid of that finding. I have seen it over and over in government agencies.

Q Do you know that for a fact or are you saying that based on your experience with public health agencies?

A Based on my experience with public health agencies and based on this paper itself. If you -- I

mean, if you tell a group of epidemiologists that that is what they did, everyone would say that is not appropriate.

My epidemiologist threw up her hands and said, I have never seen anything like this in my entire life. What are they trying to do?

Q Well, have you ever seen a published critique or criticism of the Dean paper?

A I haven't looked for one, but I am not aware of any. It was published a long time ago, 1988.

Q Has anybody gone into the St. Louis Park, Minnesota area since 1988 and tried to confirm or contradict the findings in either the Dean paper or the Dusich paper?

A Not that I am aware.

Q We will mark this next one 135.

(Defendants' Exhibits 135 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is the Eldridge paper. Eldridge is cited in on your reference list for breast cancer as number five; is that right?

A Yes.

Q And the title is Genotoxicity of Environmental Agents in Human Mammary Epithelial Cells; is that right?

A Yes. Um-hmm.
Q What is a mammary epithelial cell?
A It is a cell from the breast tissue.
Q Is it close to the outside of the breast tissue or is it closer to the skin?
A No, it's a ductal cells. It's the -- when they say epithelial, they are talking about the lining of the ducts in the breast.
Q And what did Eldridge conclude?
A Well, they were screening various agents to see which ones caused DNA changes that would be compatible with cancerous change or precancerous change.
Q And one of these agents was TCDD?
A One of those agents was TCDD. One of them was 712 dimethylphenanthrene. One was tobacco smoke, and one was benzopyrene.
Q Okay. And what did she conclude?
A Positive response is absorbed with direct acting agents suggesting that HMEC may lose their metabolic capabilities in long-term cultures.
The HMEC UDS assay will be used to address the role environmental agents in human breast cancer by determining whether chemicals are DNA reactive for metabolized and DNA reactive species in this critical target tissue.

Q This was an in vitro study?
A Yes.
Q That means that the cells were taken out of women or taken out of breast tissue. The breast tissue was --
A Reduction mammoplasty. Women who were having their -- they had normal breasts and they were having them reduced in size. So they could take out some breast tissue to do that.
Q They take the extra tissues, then, and Eldridge and her co-authors then experimented on the tissue that had been removed; is that right?
A Yes. They grew it up in a culture.
Q And then they introduced these agents to see what would happen?
A That's right.
Q And so it is not a case control study?
A No. It is a basic, you know, do these types of chemicals cause this disease.
Q And does it indicate --
A It shows relevant potency, too. I mean, some things are more powerful than others causing the effect.
Q Does it contain relative risk data for breast cancer?
A No.

Q Does it indicate a statistically significant relationship between any particular exposure and breast cancer?

A No, it doesn't. He just talks about the agents itself.

Q And the exposures that you think are relevant to our case are TCDD, benzopyrene, and what else?

A The anthracene.

Q The study states that, no UDS activity was seen with 2, 3, 7, 8-TCDD; is that right?

A Correct.

Q And so it is negative for TCDD?

A That's correct. It is positive for the PAHs. It shows the BP, benzopyrene, was a more stronger inducer of UDS than an equimolar concentration of DMBA. These data correlate with in vitro mutagenicity and DNA binding levels.

Q All right. And what is DMBA?

A That is the anthracene, the other PAH.

Q Was there an effect detected with aflatoxin?

A Yes.

Q So aflatoxin produced the result that they were looking for?

A Yes.

Q And what -- just so I am clear, what they were

looking for was a DNA repair response; is that it?

A Yeah. What was it? UDS means unscheduled repair or something or other. Unscheduled -- what is it? Unscheduled DNA synthesis.

Q And --

A Induced by chemicals. It is a marker of genotoxicity.

Q That is not surprising that TCDD did not show a genotoxic response exactly because TCDD is not a genotoxin; correct?

A Yes.

Q PAHs are?

A Yes.

Q As is aflatoxin?

A As is aflatoxin, that's correct.

Q Did they study anthracene?

A I didn't see that mentioned here. I read you the list.

Q Yeah. Next one on your list is Falck, F-A-L-C-K?

A Yes.

Q I am handing you what we have marked as Deposition Exhibit 136.

(Defendants' Exhibits 136 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is a copy of the Falck paper that you have cited; is that right?

A Yes.

Q And the title is Pesticides and Polychlorinated Biphenyl Residues in Human Breast Lipids and Their Relation to Cancer; is that correct?

A Yes.

Q And was this another in vitro study?

A No. This is a measurement of PCBs and also DDT and some other chlorinated pesticides in women mammary tissue, who had breast cancer, in 20 patients and 20 controls. So it was a human study.

Q A human case control study?

A Human -- yeah, I guess you could call it a case control. The cases were probably matched pretty well. Let's see, benign breast disease.

Q And what did Falck's --

A And they matched as close as they could on height, weight, and smoking, and no dietary history was available.

Q What did Falck, et al., conclude?

A I think that there was a correlation with PCBs and DDT and the levels were higher in the case and control; and it was statistically significantly higher.

Q So PCB and DDT. Did they study dioxins?
A No, this was PCBs using the Webb-McCall technique, as I said before.
Q And this is the technique that you thought was not reliable?
A It is reliable, but it does not measure as many PCBs because it only measures those -- the pattern -- the peaks that are similar to Aroclor 1260 or 1242. So they count all of the peaks.
They don't quantify all of the PCBs, so -- here it is. "PCBs were calculated as Aroclor
1260 (peaks with prevention
Time greater than that for p, p DDE)
By the method of Webb and McCall."
And see, that technique is not as accurate in terms of assessing the PCB body burden or the specific congeners.
So this is an older technique. And, you know, it is not going to be a good -- as good a characterization of the dioxin-like PCB.
The congener specific studies are. And -- but still, this -- they found a positive correlation. This paper triggered a whole bunch of more papers to be written and huge arguments had occurred.
Q What kind of arguments?

A Oh, other people did studies and said, well, we did not find that, so it must be wrong.

Q So there was a debate in the scientific literature about whether Falck was correct?

A There was some disagreement about it, yes. Now, I think now things are beginning to come back with the congener specific analysis. They are going to come back and probably validate what they put forward in '92.

Q What Falck, et al., put forward in '92?

A Yes.

Q But Falck and his co-authors were looking at PCBs generally, not specific congeners?

A That's right. They looked at, as I said, a summation technique.

Q And that was the technique that we saw cited in later literature where the author said it didn't show any association?

A Yes.

Q And what Falck actually does say is on page 145, "The finding of higher tissue levels among cancer cases may also signify a redistribution of chemicals to the breast during the disease process."

Do you see that?

A Yes.

Q Do you agree with that statement?
A I think subsequent events would indicate that is probably not occurring.
Q That subsequent examination of specific levels of specific congeners --
A Yes, where they looked at blood fat PCBs and -- you know, this was a tissue issue. There is no reason to think that patients with breast cancer would have higher blood PCB levels.
I mean, there is just no precedent for that. In fact, there is not even any data to support the notion that there is distribution of greater number of PCBs into the breast tissue of a patient with breast cancer. I mean, there is no biological support for that notion.
Q But why do scientists look at breast tissue and calculate PCB levels in breast cancer patients as opposed to looking at -- I don't know -- legs or toes?
A Because breast tissue is a fatty tissue and the chemical accumulates in fat.
Q Is lipid filled?
A Yes. But you are also interested in disease in that organ. You want to know is there a concentration in that organ of this chemical.
Q So Falck, et al, did not calculate relative

risk; is that correct?

A No, they didn't. All they did is 20 patients and 20 controls. It is not a population study. You can't do relative risk. What you are doing here is a biomarker study.

Q Simply showing a correlation between a level of --

A Right. As they say in the brief introduction, this class of compounds is a good candidate for being a risk factor for breast cancer. That is why they looked at it.

Q That is really all they are doing, to try to find out if it is a risk factor?

A Right.

Q Not to find out to what extent its risk factor or what dose level --

A No, there is no quantification intended or implied here.

Q And --

A And what it does simply say is that the higher the exposure, presumably the higher the risk.

Q Does it say that?

A No, I said it implies. That is the implication of the study and that is why it created such a stir, because it raised the possibility, oh, my God, there may

be this chemical that is in every single person in the United States.

It is in half the foods that we eat and it causes breast cancer, maybe that is why the rate of breast cancer has doubled in last 20 years.

Q PCBs are in half the food we eat?

A Oh, yeah, just like dioxins. Particularly in farmed salmon.

Q Right. We talked about that.

A But there is a lot of other foods where it is present not in such high amounts like in salmon, but it is present.

THE WITNESS: Time for a break?

MR. HOPP: Let's take one.

(Brief recess.)

(Defendants' Exhibits 137 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Handing you what we have marked as Exhibit 137. This is the Hansen article referenced in your report at number seven under breast cancer; correct?

A Yes.

Q This actually talks about male breast cancer after occupational exposure to gasoline and vehicular combustion products; right?

A Yes.
Q What relevance does it have to Sherrie Barnes?
A Well, that's a good question. The mechanism of breast cancer in men is possibly different than the breast cancer in women.
I mean, men and women's breast cancer may have a different etiology. I think probably the important issue here is this would be support of these chemicals that were, in this case, particularly the benzene and the PAHs are present in our case here.
And the implication of the study was that there was an increased risk that they thought was attributable to these exposures and this is a one case report.
It is not terribly important to our overall case, but it is -- let's go to the last paragraph where he discusses this issue.
He basically talks about, "The
Elevated risk of breast cancer
Among men, occupational exposed
Gasoline and combustion products
Has not been reported previously
Except in one small study with
nonsignificant odds ratio of 1.3.
However, two recent studies show an
increase in breast cancer in women

exposed to benzene and PAHs."

Which is the Petralia study, I believe is also on this list. I know it is on my new list.

And it is here on this list, and then he states, "The similarities among some of

The known risk factors for breast

Cancer in men and women and a

Similar variation in incidents

Point to common etiologic factors;

therefore, gasoline and combustion

products caused breast cancer in

Men. It probably does so in women,

too."

And then it goes on to discuss some other things.

Q So the author is hypothesizing that this result that he obtained in this paper might be applicable to women, as well; is that fair?

A Yes, and then he alludes to some other studies that showed he doesn't do an exhaustive review. Where we actually know that there are other papers that he could have cited.

Q Sure. And we will get to those.

A But the point is that it is just another study of a case of someone who has some pretty good exposures

to these chemicals that developed a rare disease.

Oftentimes rare diseases, like men's breast cancer or mesothelioma can give us a lot of clues and should be followed up when they occur.

Q Would it be fair to characterize the Hansen paper as generally informative, but not directly related to the cause of Sherrie Barnes' breast cancer?

A Yes.

Q In fact, the article does not calculate the relative risk for breast cancer in women; is that right?

A Correct.

Q And at what exposure level does the study indicate that breast cancer has increased in men?

A Well, he has got an odds ratio here of 2.2 with no lag time and 2.5 with ten years of lag time with statistical significance.

Q Lag time being years of exposure? What does lag time mean?

A No. What that does is it allows for more latency.

Q Okay.

A In other words, you look at the people's exposure and then you make sure that you are at least allowing for ten years of lag time from the time of exposure to the time of the disease diagnosis.

Q Is it accurate to say that the Hansen study doesn't examine specific exposure levels, but rather looks at occupational exposure of gasoline and combustible products in general?

A Yeah, 230 male employees were members of the National Pension Fund and the country is Denmark. And he looks at job title for exposure.

Q Okay. So there is no exposure data for the individual study subject?

A No.

Q Next one on your list -- your breast cancer reference list number eight is the Holford, H-O-L-F-O-R-D, study?

A Yes.

Q Handing you what we have marked as Deposition Exhibit No. 138. This is a copy of the Holford study. The Holford study is entitled Joint Effects of Nine Polychlorinated Biphenyl (PCB) Congeners on Breast Cancer Risk; is that right?

(Defendants' Exhibits 138 was marked for identification by the court reporter.)

THE WITNESS: Yes.

BY MR. HOPP:

Q And Holford looked at nine PCB congeners; right?

A Yes.
Q And, generally, what did Holford conclude?
A There is an association with some of them. Let's see if I can make sense out of this.
Table 2 shows odds relative risk associated with a ten PB change in exposure to individual congeners by type of model; and I think the risk associated congener values that are listed in the middle .2153 and 156 is not being significant.
Q Okay.
A But 183 is significant.
Q 180 is slightly elevated, but not significant; right?
A Yes, 180 is slightly elevated, but it is not very significant. It is almost significant, it is .99. It is real close. Anyway --
Q But 183 is the culprit in that Holford paper; right?
A That is the one that they felt was statistically significantly associated.
Now, on Table 3, they give an odds ratio associated with a level of PCB in quintiles and they divided them into five levels.
Q I'm sorry. Table 3?
A Table 3, it is at the bottom of 979.

Q Okay.

A And there, they -- as the level of the PCB increased, the odds ratio of relative risk -- I think it is related risk score, which is similar to relative risk, it is adjusted estimates of relative risk. Risk becomes statistically significant only at the top quintile. Otherwise, the curve is pretty flat.

Q And Table 3 is looking at all of the congeners that are being studied or is it --

A They have some kind of PCB score. It is a score -- let's see how they scored it. Somewhere in here they describe the score.

All right. Well, it is on Page 977. It is called Principal Components, and they describe what they did.

"In order to understand better the nature of the effects for individual congeners, principal components analysis was used to create factors that were independent of each other. Using PRO PRINCOMP in SAS we estimated the eigenvectors, which provided loading scores that gave rise to new variables to be

included in linear logistic model."

Q What the heck does that mean? Do you understand that?

A Yeah, they are doing statistical analysis, which is -- when you have multiple variables like this, you know, a dozen or so PCBs, plus other variables, age and whatever else you put in the model, you have got a very complex statistics; but not being a statistician, I cannot really explain to you what they are doing. It is a very high order statistical.

Q Well, principal component analysis is the general name for what they did?

A Yes. Well, that component.

Q All right.

A In the Statistical Methods, they discuss their analysis, how they did it, and one they want to look at the joint effects of individual PCB congeners on the risk of breast cancer and whether the effect of each congener was the same, which was tested using linear contrast.

"If these results suggested
That the magnitude of effect on
Breast cancer risk was different
From the congeners, then it
Would not make sense to evaluate

Total PCB exposure, but to
Investigate the joint effects of each
congener. Regression diagnostics
Were used to determine whether
The results were sensitive to one or
more influential observations."

Q I'm sorry.

A Now, we are talking about regression diagnostics was used on one or more influential observation.

"But the overall conclusions
Were found to be stable. Bootstrap
methods were used to estimate
Bias in the estimates of risk, as well
as providing alternative estimates of
standard errors. While the resulting
standard errors were slightly greater,
the conclusions were essentially
unchanged, so these results are not
present."

I think what they are saying is that their principal component analysis is what they used and that is what they used in Table 3 as a related risk score.

Q And above Table 2, the authors point out, the statement is, "Notice that some congeners

Are positively associated with breast cancer risk, while others are negative"; is that right?

A Well, if you look at the standard coefficient, the first line, when it says, negative .021, that means that the higher the PCB level of the congener, the lower the breast cancer risk.

Q All right.

A So that is right. There were three -- four that were negative and then one, two, three, four, five that were positive.

And 180 was the most positive statistical and it reached almost statistical significance and 183 did.

Q And in the Discussion section, this is on Page 979, the authors point out that, "The Association of total PCB exposure with breast cancer risk in this analysis was estimated to be small and inverted."

Is that what you are talking about?

A Yes, for those who had it -- the higher the level, the lower the risk, suggesting -- I think, you know, you can find and do these fancy statistics. You can find things like this. That may not mean anything.

The most important thing here is to look at all the congener correlations, and 180 and 183, again,

correlates strongly with the total congeners.

In other words, you are getting a positive effect on the breast cancer. And like the other studies we have looked at, if you add up all of the PCB congeners, that also correlates with breast cancer risk.

So what it would suggest is that the overall mixture, maybe some components being more important than others, is contributing to the risk; and that the negative components do not outweigh the positive components in terms of causing the effect that we are seeing in the increased risk.

Q But they do balance out and that is why the authors say that the overall risk is small?

A That's correct.

Q And they go on in the Discussion section and say, "These results suggest that some Congeners have a protective effect on breast cancer risk, while others are associated with an increased risk"; is that right?

A That's right. That is correct.

And I think that is consistent with all the data. It shows that there is a small but significant increase in risk. And the reason it is important is that there are so many darn people exposed and so many

people get this disease, that anything that contributes to the risk is important to address.

Q Is this a case control study?

A This is a biomarker study. I mean, there is cases and controls. What they are doing is they are studying the presence of a biomarker in PCBs in two populations to see if the testing hypothesis that the cases would have a higher level of these chemicals than the controls.

And the answer is, yes, and it does show correlation.

Q Does this study indicate what dose of any particular PCB congener is necessary to cause an increased risk of breast cancer?

A No, I mean, if you look at the -- I don't think there is a single measurement in this whole paper. It is all statistical analysis.

Let me just see. Maybe they are mentioned somewhere. The level -- no, what they are really trying to do is the correlation or the association of the chemical versus the risk. And that is not going to give you thresholds or slope factors.

Q The next paper in order on your reference list, this is number nine, is the Hoyer paper; is that right?

A Yes.

Q I am handing you what we have marked as deposition Exhibit No. 139.

(Defendants' Exhibits 139 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Is this the Hoyer paper?

A Yes.

Q And it is entitled Organochlorine Exposure and Risk of Breast Cancer. What question was Hoyer trying to answer?

A The same question. He looked at Dieldren, which is an organochlorine. He looked at chlorocyclohexane, which is another pesticide, organochlorine pesticide.

Q Did this study look particularly at TCDD or dioxin?

A No, it looked at PCBs, DDE, but it did not look at dioxin per se.

Q So this would be another study that is generally informative, but it is not directly related to Sherrie Barnes; is that right?

A Yes. For the reasons that I indicated earlier, I thought it was relevant.

Q And they actually looked at serum levels; is that correct?

A Yes.
Q So these are blood samples and not tissue samples?
A Yes, serum sampling. That is right.
Q The Result section indicates that,
"The risk of breast cancer decreased with increasing number of full-term pregnancies and increased with" -- I'm sorry -- "and increasing with body weight and height."
Do you see that?
A Where are you reading from?
Q The Result section, this is Page 1818 starting right above that table.
A "Increasing number of full-term Pregnancies and increasing with Body weight and height."
So height was made a standard.
Q You wouldn't think so. But Hoyer at least concludes that increasing body weight and height are a risk factor; is that right?
A This is the first time I have ever seen height as a risk factor for anything. And unmarried women had an 89 percent higher risk than married women. It is probably because they didn't have babies.

Q Moving on down this page, this is 1818. It says, "We found a slight increase in
Risk of breast cancer with increasing concentrations of BHCH, but no association was apparent for total DDT or total PCBs."
Do you see that?
A Um-hmm.
Q So this study tends to conflict with some of the other studies which have indicated PCBs increase the risk of breast cancer?
A Well, they did 28 PCBs. They don't tell us which ones. So this wasn't as detailed a congener analysis as the others.
They do list them here. And -- yes, they just didn't find a correlation.
Q And then the Conclusion, which is on the last page states, "Our results support the
Hypothesis that organochlorine compounds, such as dieldrin,
Which have oestrogenic properties,
May increase the risk of breast cancer.
They do not, however, suggest that exposure to total PCB, total DDT,"
And I guess, "P prime-DDE have any

influence on the risk of breast cancer."
Is that right?
A Yes, in this study, they did not find an increase in breast cancer. That's correct.
Q The next study in order under List of Breast Cancer References is the -- maybe you can pronounce it for me. Kogevinas paper?
A Kogevinas is as good as any.
Q Kogevinas, K-O-G-E-V-I-N-A-S.
I am handing you what we have marked as deposition Exhibit No. 140, which is the Kogevinas paper.
(Defendants' Exhibits 140 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Now, this is a review article; is that right?
A It is.
Q So it doesn't report on a new experiment, but rather discusses studies done by other people?
A Yes.
Q And does Kogevinas find -- well, let me -- what does Kogevinas conclude, generally, based on the other studies?
A More studies are needed. That was his main conclusion, but he reviews some of the studies and it is

interesting in that respect.

Q And that is, again, generally informative, but not particularly relevant to Sherrie Barnes?

A Correct. He gives a list of the various studies and notes, you know, the breast cancer, including male breast cancer, has been found to be increased.

Q He finds increasing mortality from breast cancer that is not statistically significant; is that right?

A Yes.

Q That is in Table 5?

A Yes. Table 5 he is looking at -- where is that? He has got different references 170 -- where is it? I am trying to see what his references are for that.

Anyway, he -- I guess, IARC's international cohort study of phenoxy herbicides or chlorophenols where TCDD was presumed to be present and the SMRs are elevated for all of the cancers, but all malignant neoplasms are statistically significantly increased.

And the individual types of cancer, breast female is almost statistically significant. The odds of SMR is 2.16, but the confidence interval is at .99. We are talking about 100ths off. Otherwise, it would be

statistically significant.

So that, in light of all the other evidence we have, this is supportive.

Q All right. But you are looking in this paper, Table 5, you are looking at nine deaths; is that right?

A Yes.

Q Out of how many expected?

A Well, that would be 2.16 more than expected. So you would expect in that population -- I guess, the 174 reflects the number of something rather -- what is it? I don't know the number of people at risk; but they expected half of that many cases. So there is a doubling of risk.

Q If the spread at the 95 percent confidence interval includes one, then it is not statistically significant?

A Yeah, I know. And if it was one more, it would be.

That is the point I am trying to make is it is very close to statistically significant; but if the numbers were bigger, it would be significantly.

And as I say, by itself, it would not be important, but taken in light of all of the other evidence, it is supportive.

Now, the same is true of male breast cancer.

It has doubled 2 1/2 times the -- twice as they expected.

And, again, that goes along with our other observations about this and, similarly, prostrate is elevated. Testes is elevated. Thyroid is elevated, and all endocrine organs are elevated. The numbers are small.

Q And not statistically significant?

A Not statistically significant, but the point is all of these cancers are endocrine disruption sensitive cancers. And, again, in view of other information, it certainly is worth paying attention to.

Now, if you go over to the last one,

"All workers exposed to any phenoxy herbicide or chlorophenyl."

Q Still on Table 5; right?

A Still on Table 5. You have got a statistically significant excess of, again, all malignant neoplasms and other endocrine organ cancers are elevated statistically significant.

So it would appear to me that, you know, this paper is useful.

Q In a general way?

A Correct.

Q It does not identify a particular dose level

which is required to increase the risk of breast cancer; is that correct?

A No.

Q The next paper in order on your list of breast cancer references is the Laden paper, L-A-D-E-N; is that right?

A Yes.

Q And it is number 11; correct?

A Yes.

Q I am handing you what I have marked as Deposition Exhibit No. 141.

(Defendants' Exhibits 141 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is the Laden paper; is that right?

A Yes.

Q And the Laden paper looks at the Nurses' Health Study; is that right?

A Yes.

Q Is that otherwise sometimes called the Harvard Nurses' Study?

A Well, this is from Harvard. So it could be considered the Harvard Nurses' Study.

Q Have you heard that expression before, the Harvard Nurses' Study?

A No. I heard the Harvard Doctors' Study, but yesterday when you mentioned the Harvard Nurses, this is the first I heard of it.

But as I said, this is a study of nurses conducted by Harvard. So it would be appropriately called that.

Q And correct me if I am wrong, but it appears that what happened was Harvard or some group at Harvard has collected and has continued to collect data on a large group of nurses.

It is sort of a prospective study. It examines health effects over the course of the lives of these women?

A Yes, just like the doctors' study. Same idea.

Q The idea is to --

A Follow the large group and see what happens to them and look at the different risk factors prospectively.

Q It states, at the end of the abstract,

"The majority of studies have concluded the exposure to PCB are unlikely to be a major risk factor for breast cancer."

Is that right?

A Are you talking about --

Q I am looking at the end of the abstract.

A Although there is no independent association, blah, blah, blah -- yeah, the point of this paper is that if you look at the nurses who have this particular polymorphism, CYP1A1-exon 7, this is a risk factor for breast cancer.

Q Okay.

A And I think what they found was --

Q Was what he found that this was a genetically susceptible population?

A Correct.

Q Okay. Doctor, do you want to continue with your answer?

A What they say here is, "However

High levels of PCBs may be associated with breast cancer risk in the subgroup of women who have variant CYP1A1-exon 7 polymorphism."

Additional studies are needed to examine that possibility.

Q That is CYP1A1-exon 7 polymorphism, that is something to do with the particular genetic structure of these women; is that right?

A Yes.

Q It is a gene?

A Their ability to transform the PCBs or handle

them is impaired or reduced.

Q And that's the only study -- strike that.

That is the only population in which the Laden paper found an effect with increased levels of PCB; is that right?

A That's right.

Q And we don't know whether Sherrie Barnes had that particular polymorphism, do we?

A No. You asked me that yesterday. So we don't have any studies on Sherrie Barnes or anybody else on cohort. It is not a routine thing you send to the lab.

Q You have to take a tissue sample?

A You have to do genetic studies. That is what you have to do to find this particular variant. It is expensive and it is possible to be done. But it is very important particularly in people that we don't have disease in yet; but we want to know who is at high risk. These kind of studies would be highly relevant.

Q Would it be possible to test Kenesha Barnes to find out whether her mother had that particular polymorphism?

A Well, we would have to check her dad, too. I don't know how the inheritance goes for that particular gene. I don't know if it can be an acquired defect. I would have to study it to answer that question whether

or not it would be relevant to test her.

Q This study looks at latent PCBs; correct?

A Correct.

Q Doctor, I have handed you what we have marked as Exhibit 142. This is the next reference on your breast cancer list. It is number 12 and the author is Leis or Lees. L-E-I-S.

(Defendants' Exhibits 142 was marked for identification by the court reporter.)

THE WITNESS: Yeah.

BY MR. HOPP:

Q And this is really just a paper on diagnosing breast cancer; is that right?

A Yes, it has risk factors. That is the reason it is here.

Q But does it talk about environmental risk factors or TCDD?

A Not really, it talks -- Table 1 and Table 2, exogenous estrogen, which would be in birth control pills and hormone replacement. And then it says,

"Carcinogenic exposure,
particularly to viral agents
and some drugs."

Q So --

A Really, it just kind of gives you a list of

things that have been raised as -- just kind of a general review of the disease. So you know what you are talking about.

Q Not very informative with respect to causation?

A Correct. I don't think he has references for a lot of those causative factors. He doesn't give a reference. He makes the assertion in this table.

Q The next breast cancer reference that you have in order, number 13, Lucena, L-U-C-E-N-A; is that right?

A Right.

Q I am handing you what we have marked as Exhibit 143.

(Defendants' Exhibits 143 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is the Lucena paper; is that right?

A Yes.

Q It is entitled Short Communication. Is this -- is there some significance to that?

A Well, what they do is they write a very brief paper presenting one table, maybe, which they think is important when they want to publish it as a -- quickly, so it is easier for the reviewers to deal with a short paper with very little information, so you can get it published faster.

Q Right. And this paper really identifies one specific congener or PCB?

A 28.

Q As associated with an increase risk of cancer; is that right?

A Yes. Fascinating. It is like every paper has a different congener. Congener of the week.

Q Did Lucena look at other congeners?

A They looked at a bunch of them. It is listed on the top of 118, left-hand column.

Q But the only one they found that significantly increased the risk of breast cancer was 28; correct?

A Yes.

Q Once again, they think there is a great need for more studies?

A That's, as I told you, every study will say that. It is the stock and trade of a researcher.

Q Does Lucena calculate a relative risk for exposure to PCB 28?

A Yes, 9.597, huge odds ratio. Same thing.

Q But it does not identify a particular dose level for that congener which would result in that increase risk; is that correct?

A I don't see that it was quantified. What they said was in the difference between the exposed and the

controls, it was a ninefold difference in that chemical.

Q So what they were -- this was a study in Spain; is that right?

A Yes.

Q And they were actually looking at breast tissue that had been removed from women who had breast cancer; correct?

A Yes, that's correct.

Q And these were malignant lesions?

A Well, in the exposed, they were malignant.

Q And the controls, they were benign lesions; is that right?

A Benign lesions.

Q So what they found was that if someone had a detectible level of PCB 28 in the malignant lesion, those people turned out to have a 9.597 odds ratio; is that correct?

A That's right.

Q How is this paper -- strike that.

How does this paper relate to or inform your opinion with respect to Sherrie Barnes?

A The same as the other PCB papers. We are talking about a similar toxicity for dioxin-like chemicals.

Q The next one in order on your breast cancer

reference list is the Manz paper; is that right, M-A-N-Z.

A Yes.

Q I have marked that deposition as 144. This is the Manz paper; is that correct?

(Defendants' Exhibits 144 was marked for identification by the court reporter.)

THE WITNESS: Manz paper, correct.

BY MR. HOPP:

Q M-A-N-Z?

A M-A-N-Z, from Germany.

Q This is a German study of exposure, actually, of workers in a chemical plant; is that right?

A That's correct.

Q And they characterized -- first of all, it is a retrospective mortality study; correct?

A Yes.

Q And they characterized the herbicide workers in this plant in Germany as being having been exposed to heavy contamination of 2, 3, 7, 8-TCDD?

A Yes.

Q But only seven percent of the women worked in high exposure areas of the plant; is that right?

A Yes.

Q Did they detect an increased risk of breast

cancer as a resulted of heavy exposure of 2, 3, 7, 8-TCDD?

A I think this is overall, the SMR for carcinoma of the breast was 2.15 with a 95 percent confidence interval of 0.98.

Again, right at the borderline, and 409 for nine deaths.

Q And this is what table?

A It is on Page 962 under Mortality Among Women.

Q All right.

A Malignant neoplasms were right at not significant, but the breast cancer was. And that's really the point of the paper, which is about TCDD.

Q Okay. So it is just about TCDD, and the 2.15 is an increased SMR, but is it statistically significant?

A Well, it is right at that borderline at 0.98.

Q Again, the 95 percent confidence level includes one?

A That's right. It is right at the borderline.

Again, I think I have said it before, when it is taken into the context of everything else, it is supportive. They also review a study, which I don't think we got, but --

Q Which study is that?

A I am just looking at it here. I am wrong. That's the only point of this study.

Q Does the Manz paper identify a dose level of 2, 3, 7, 8-TCDD, which is significant for increasing the risk of breast cancer?

A No.

Q Is the exposure level documented in the Manz paper?

A No, they don't do blood levels or the chemical plant was found to have high TCDD levels enough to cause chloracne. And that was led to the change in practices to reduce exposures.

Q So qualitatively, they think it was high because of the chloracne?

A Well, we know that when you get chloracne, you are at high levels; but they don't give the numbers in here.

Q While we are on the subject of chloracne, I know I discussed this with Dr. Sawyer, and forgive me if I covered this with you.

Are you familiar with the case of Victor Yushchenko?

A Yes, I am.

Q Victor Yushchenko is the president of the Ukraine; is that right?

A Yes, he is.
Q He was actually -- someone tried to poison him with dioxin?
A That's right.
Q Do we know if it was 2, 3, 7, 8-TCDD?
A No, we don't know precisely, but he had dioxin poisoning. And in the poisoning episode, they usually use TCDD because it is available. If you are running a lab when you are testing this, you can get TCDD as a standard.
Q You could have gone to the German factory and seen the Manz paper and gotten it?
A Yeah, I guess so. You can get purified TCDD from a chemical supply house.
Q Now, the acute exposure to -- strike that.
I believe Dr. Sawyer testified that the level of Victor Yushchenko's exposure to TCDD was among the highest ever recorded?
A Among the highest recorded, that is correct.
Q There were a couple of other acute poisoning cases that were documented, several women 20 years ago or so, who were up in that range, as well; is that right?
A Yes, from Vienna, Austria.
Q But Victor Yushchenko did not die from his

poisoning; correct?

A Not yet.

Q How about the women in Vienna, Austria; did they --

A They have not died yet either, but they are being followed. They are about ten years from the onset of exposure. And --

Q Did they --

A They are quite ill and I suspect Victor Yushchenko is quite ill. They have been attempting to get the levels down using various techniques to detoxify, but nothing is working. But the levels of both the two women from Austria and Yushchenko are still extremely high.

Q They were using Olestra, I think, with Yushchenko; is that right?

A They used Olestra with the two ladies from Vienna, also.

Q Did it work?

A It is a miserable, miserable drug. It causes diarrhea and people can't take it. So they take it for a while till they get sick of it. It may lower the level a bit. It is not terribly effective.

Q Olestra is the fake fat; right?

A That's right. The non-absorbable fat. It is

the same as Cholestyramine and the cold pressed oils that we use. It compresses (phonetic) in the gut.

Q And the women from Vienna, have they developed breast cancer?

A No, not yet. And we talked about this earlier, it may not be TCDD in the adult that causes the breast cancer, anyhow. Or it may not be nearly as potent a factor in the equation. I mean, you can induce --

Q Let's move on. The next paper you have cited in your breast cancer references, it is number 15, the Morris paper; is that correct?

A That's right.

Q I am handing you what I have marked as Deposition Exhibit No. 145. This is the Morris paper; correct?

(Defendants' Exhibits 145 was marked for identification by the court reporter.)

THE WITNESS: Yes.

BY MR. HOPP:

Q And this paper is -- would it be accurate to call the Morris paper a hypothesis-generating paper?

A Well, he reviews all the data. That's the value of reading a paper like this.

Q What, if anything, does Morris conclude?

A Well, he talks about benzene, benzopyrene. He

does talk about cigarettes, aromatic hydrocarbons, and breast cancer, and PAHs.

Q Is Morris a review paper?

A Yes. He goes on to talk about PAHs and in quite a bit of detail. And then concludes, you know, that something going on in our environment is causing this. And his candidate is aromatic hydrocarbons, in a broad sense.

And he reviews a bunch of them. And, of course, PAH is at the top of the list here. He does not go into much detail on the polychlorinated hydrocarbons.

He is mainly focused on the aromatic hydrocarbons. It is a very thorough review of those papers up to that time.

Q It is sort of a dated paper; right, this is '92?

A '92, but there was still quite a bit more evidence already at that time.

Q Morris identifies radiation and aromatic hydrocarbons as inducing and promoting mammary cancer; is that correct?

A Yes, that's correct.

Q He also states that such disparate factors as urban residents, geographic location of residents, and life-style factors, such as alcohol ingestion, high

polyunsaturated fat diet, and food selection and preparation all contribute to exposure to promoter and initiating influence of aromatic hydrocarbon carcinogenesis; is that right?

A That's correct. That is what he says.

Q Does Morris isolate any particular exposure, any particular PAH which he thinks is significant for causing breast cancer?

A Benzopyrene and DB(AH)A anthracene, which are the experimental animal carcinogens. He also mentioned DMBA and PAHs in general.

Q In his review, does he discuss human epidemiology studies, or any the animal studies and in vitro studies?

A Well, he does -- he touches on animal studies quite a bit. Because in '92, there were fewer studies, but he mentions benzene, as well, and its ability to induce cancer, and talks about the -- mostly the animal study.

There wasn't as many studies back at that time in humans as there are now. But he gives a background as to why people started looking so hard at human studies, subsequently.

And he points out why these chemical PAHs, in particular, are likely to be the cause of breast cancer.

Q He does not address creosote as a mixture; correct?

A No, he doesn't.

Q And does he document any exposure levels to any particular PAHs?

A No.

Q Does he calculate relative risk levels?

A No, he doesn't do that either. This is a review paper of pointing out all of the papers that exist at that time that point towards a link between the PAHs and breast cancer.

Q I understand, but Morris does not identify any particular exposure level that is necessary to produce harm; correct?

A No.

Q I'm sorry. That was a bad question.

Does Morris identify a particular exposure level that is necessary to produce harm?

A No, he doesn't.

Q The next paper on your list of breast cancer references is number 16, Muscat, M-U-S-C-A-T; correct?

A Yes.

Q I am handing you what we have marked as Deposition No. 146.

(Defendants' Exhibits 146 was marked for

identification by the court reporter.)
BY MR. HOPP:
Q This is the Muscat paper; correct?
A Yes, it is.
Q Entitled Adipose Concentrations of Organochlorine Compounds and Breast Cancer Recurrence in Long Island, New York; right?
A Yes.
Q So, again, he is looking at PCBs; right?
A Yes.
Q And what, if anything, does Morris conclude?
A Muscat.
Q I'm sorry. Muscat conclude?
A That there is a linkage between adipose PCB levels, which is -- let me see. I think it is recurrence in -- of breast cancer.
Q Muscat is looking at cancer coming back a second time?
A Yes, he is talking about it being a predictor of recurrence of breast cancer. Interesting study.
Q How does this relate to Sherrie Barnes?
A Again, it is showing PCBs which are dioxin-like in their behavior increasing the risk of recurrent cancer, which is relevant to our patient, I believe, in the sense that she had a tumor that was very aggressive.

What they are suggesting here is that PCBs probably increased breast cancer risk, but that is not the main point. The main point is that it is -- or associated with recurrence.

Q So Sherrie Barnes had it once and it was fatal?

A Yes. She did not have a recurrence. She did not respond to the therapy either, suggesting that her tumor was very aggressive and malignant.

And this paper suggests that making the tumor grow more readily would be associated with these types of exposures.

Q And in the concluding paragraph, they point out that these results, that is, the results represented in deposition Exhibit 146, conflict -- I'm sorry -- contrast with the author's previous data showing no effect of organochlorine compounds of breast cancer in these women; is that right?

A Yes.

Q So there was a previous paper by the same authors which was negative; correct?

A That's right.

Q Does the Muscat paper calculate relative risk of recurrence?

A Let's see, relative risk is on Table 5, and as the level grows, most of the relative risk grows

significantly with each PCB congener.

It is interesting. Not all of them were that way, but starting with -- with the 74, lowest tertile was 1; middle tertile was 1.3; and highest tertile 1.7.

And then, anyway, they go all the way down. Some of them are statistically significant. Some aren't. The total PCBs is most significant at the highest tertile. 2.9 is the relative risk with the statistical significance.

Q This paper actually does contrast with some of the other papers we looked at, even today, which show that some of these same congeners do not increase the risk of breast cancer; correct?

A Yeah, I think it would be -- they need bigger numbers, probably, to do that, but more importantly --

Q Explain that. Who would need bigger numbers to do what?

A Well, how many patients did they have? 30 patients in the recurrence category.

Q You are talking about Muscat?

A Muscat. If you had maybe 300, you might be able to start seeing differences in the individual congeners; but they do have mean concentrations in the blood of the various congeners and consistently -- pretty consistently, they are higher in the recurrence

patients all the way -- there are three or four that aren't.

And no one stands out, but then the total turns out to be statistically significant. And the biggest difference is in the blacks. Where they -- it is 129 parts per billion difference.

Q Can you explain that? What does it say about black women?

A That blacks with no recurrence, their PCB total was 406. The blacks with recurrence, their PCB level was 529. Both of those values were higher than the whites.

And the highest at all are the Asian with no recurrence, but there is very small number of Asian, so --

Q So does the Muscat paper identify an exposure level as necessary to cause harm?

A No, they do not.

Q Muscat does indicate on, Page 1477, that there were relatively few events in this study and the positive findings could have been due to bias?

A Sure.

Q What is bias in this context?

A Something that is causing the results that is not a true cause. Bias just means that there is

something that is screwing it up.

Epidemiology people always say those sorts of things. It is just terms of epidemiology.

It could have been through chance. It could have been bias. We don't know. We tried to remove all the bias; but there is always a risk. Something that we didn't control for.

Q Isn't that what epidemiologists spend most of their time doing? Try to eliminate possibility of their chances, influencing their --

A Yes, they spend a lot of time.

Q That is the whole point. If the result is dictated by chance, then you have wasted your time doing your --

A Exactly. You are going to get a negative study. That is why they tighten, over the years, the criteria to say significant.

It used to be, when I started out in medicine, P value of .1 was considered significant. Now, it is .05. So you have to have a really good study, really a strong effect to get statistical significance.

Q Your next study on your list of breast cancer references is the Negri, N-E-G-R-I, study; is that right?

A Yes.

Q Handing you what I have marked as Exhibit 147.
(Defendants' Exhibits 147 was marked for identification by the court reporter.)
BY MR. HOPP:
Q This is the Negri study; right?
A Um-hmm.
Q This is a review article; right?
A It is.
Q And it looks at exposure to PCB and breast cancer?
A Yes.
Q And what does Negri and/or her coauthor conclude?
A Well, I think the important point is that you need to take into account genetic susceptibility in order to explain what is going on; and that in the general population, without the genetic risk factor, there probably isn't an increased risk.
Q So, in fact, at the concluding part of the study, right above the acknowledgments, Negri and coauthors say, "In conclusion, the epidemiological evidence does not support the hypothesis of a direct relation between environmental exposure to PCB

adulthood in the general population and the risk of breast cancer"; right?

A That is what he said in the abstract, which I just read to you.

Q And then he goes and talks about a specific genetic variation like --

A Right. He is really just repeating what we said earlier about the CYP1A1 and the exon 7. He does not mention exon 7, but in Table 5, he mentioned that.

Q But for the general public, Negri is, essentially, a negative paper; right?

A Yes, that's the point. But when you take into account the -- see, there is a couple of papers that we have not gone through that are reviewed here that make the same point.

Interaction between PCB and the CYP1A1 polymorphism, I think what the science has evolved to the point that it takes -- you can have the CYP1A1 gene and not get breast cancer; but if you have it and are exposed to PCBs, then your risk of breast cancer increases significantly.

Q And how is this study relevant to Sherrie Barnes?

A Well, it is like all of the others. I have referred to in the PCB literature. It shows the effect

of the related compound.

Q So it is generally informative, but not directly related?

A Correct.

Q The next paper in order on your breast cancer reference list is Petralia; is that right?

A Yes.

Q It is Petralia, 1999?

A Yes.

Q Petralia has written several articles on this subject; right?

A I have Petralia -- another one of the Petralia papers on the new -- that I gave you. Two more, '95 and '98.

Q So you have got some older papers?

A '98 and '99. So I have got the '99 paper, but I have got an earlier '98 paper that I have added.

Q Let me show you Exhibit 148.

(Defendants' Exhibits 148 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is 1999 Petralia paper?

A Yes.

Q And Petralia is looking at the premenopausal -- I'm sorry, risk of premenopausal breast cancer in

association with occupational exposure to polycyclic aromatic hydrocarbons and benzene; is that right?

A Yes.

Q So this is an occupational study?

A Yes.

Q And does it look at women particularly in these occupations?

A It has to be.

Q Premenopausal --

A The rate in men, as we know, is quite low. So it is women. And the exposures were variable.

They took occupational history of the exposure assessment for PAHs and benzene was developed to determine which occupations had exposure. And then they developed a matrix for that, which included the PAHs and the benzene and others things, as well.

Q And which exposure levels did they find to be significant to increase the risk of premenopausal in breast cancer in their occupation when exposed to both?

A PAH and benzene, highest risk was in PAH and benzene together. They found statistical significance in all of them and the biggest abnormalities were in the ER positive cases.

Q What is that?

A Estrogen receptor positive, which we looked at

yesterday in our case.

Q Oh, ER positive breast tumors, that is a particular type of tumor?

A Yes, this is the first time we have seen that.

Q Okay. Seen what? Seen a study?

A Seen a study where they looked at the ER positive and ER negative.

Q Forgive me for covering this again, but we don't know whether Ms. Barnes had a ER positive or ER negative breast; right?

A Yes, I don't think we do.

Q Again, forgive me for asking you to say this again, but what was the dose level that the authors of the Petralia paper found to be significant for inducing breast cancer? Did you say it was every dose?

A Well, they have got some duration data here which would be a surrogate for dose.

Q Oh, I see. They use job exposure matrixes and lifetime occupational history; is that right?

A Yes. And they had low exposure and medium to high and then cumulative low, medium to high and, in general, I think they only found a few cases that were a statistically significant; but the numbers in each cell are so small that it is not likely to find statistical significance.

So there were some elevated odd ratios. In fact, lots of them were elevated; but it didn't reach statistical significance, except in a few cases.

And, again, if you look at the ends, that is the problem. There aren't enough in each of the cells to reach statistical significance.

Q So it is too small a study, really, to effectively evaluate statistically significant association?

A With dose. It is a large enough study to say in general. I mean, you have got quite a few people in the exposed categories.

Q So overall, looking at overall exposure, they find an increase risk?

A That's right.

Q But they cannot break that down by exposure classification?

A Right. Of the patients that they looked at, they had 25 of PAH alone; 35 of benzene alone; 6 exclusively PAH; 19 PAH and benzene; and 16 exclusively with benzene. Those are --

Q Small numbers?

A Relatively small numbers, but big enough to get statistical significance on many of these; but then when the numbers drop down to 16, 8, 13, and 11, 10 and 9 --

10 and 8, then they don't get statistical significance even though they have elevated odds ratios.

Q Looking at Page 220, this is the first full paragraph. It says, "When our results are interpreted, several issues need to be considered. The response rates for both the cases and referents" -- that is R-E-F-E-R-E-N-T-S -- "were low."

Now, that is a problem for epidemiology?

A Where are you reading from?

Q Page 220, first full paragraph, starting with the words, "When our results are interpreted."

A Oh, I see, on the right-hand side.

Q Low response rate is a problem for an epi study; right?

A Yes, the response rates were low. That is a problem.

Q And would you characterize it as a case control study or a cohort study?

A Case control.

Q How does this paper, the Petralia paper, relate to Sherrie Barnes?

A She was exposed to both benzene and to PAHs, and so it would be a direct relationship. Although it

was not occupational exposure, she had environmental exposure. I would submit that she probably had higher exposures to PAHs and benzene than the people in this study.

Q Does the Petralia study, then, identify an exposure level that is necessary to cause harm?

A No, it doesn't have any quantitative data.

Q All right. The next study in order on your list of breast cancer references is Pliskova; is that right?

A Yes.

Q P-L-I-S-K-O-V-A. I am handing you what I have marked as deposition Exhibit 149. This is the Pliskova paper; correct?

(Defendants' Exhibits 149 was marked for identification by the court reporter.)

THE WITNESS: Yes.

BY MR. HOPP:

Q I have actually handed you two things. One is the abstract and one is the article.

A Oh, yeah.

Q Let me have the abstract back, so we don't confuse ourselves.

A Okay.

Q So Deposition Exhibit 149 is the Pliskova

article; correct?

A Yes.

Q And Pliskova article states what, specifically?

A Benzopyrene and -- what is the second one? Benzanthracene.

Q Is this an in vitro study?

A This is an in vitro study.

Q So, again, they are studying cells in a petre dish?

A Yes.

Q What does Pliskova conclude, if anything?

A Well, it is a mechanism paper. They talk about how it induces -- benzopyrene induces P53 tumor suppressor expression and abolish both S-phase arrest and apoptosis induced by the PAHs.

Potentiated deprecative effect of BaP. Thus, specific genotoxic and non-genotoxic event for interacting on the effects of BaP cell proliferation.

Q How about in layman's term, what are we looking at?

A I think the reason that I thought this was important is because of this notation about non-genotoxic mechanisms which hadn't been talked about too much on any other paper.

Q Well --

A And they also talk about the BAP and TCDD have some similar toxicities.

On Page 254, in the right-hand column, in the first full paragraph, about halfway down it says,

"Using a combination of DNA staining and detection of BrdU incorporation, we found that like TCDD, BAP and BAA also partially inhibited induction of S-phase entry by E2. However, unlike TCDD, both BaP and BaA also stimulated G1-S-phase transition, when applied to serum-starved cells, albeit to a lesser extent than E2 itself. Interestingly Dibeno[a,h]anthracene, a strong AhR ligand, which has been shown to be antiestrogenic in MCF-7 cells, had the same effect as TCDD both on the E2-treated and untreated cells. These results seem to support the hypothesis that unlike other PAH's, BaP and BaA, or their metabolites that are less efficient inducers of AhR-mediated activity, can activate ER

and stimulate cell proliferation."

Point being, that they have similar toxicity of TCDD and that the effects of the two together are going to be at least additive.

Q Similar effects, when they contact the Ah receptor; right; that is what they are saying?

A No, this is talking about other effects. That is the point I was trying to make.

Non-Ah receptor stimulated toxicity, because these other types of toxicity to the cells are not related to the age receptor. And they are pointing that out. That's all.

Q Let's look at the first page of the article. This is on the right-hand column, about an inch or so down, she says, "Today, PAHs are

regarded mostly as antiestrogens principally due to their ability to activate aryl," A-R-Y-L, "hydrocarbon receptor," that is AhR receptor, "which may lead to supression of estrogen response element controlled gene expression."

So they are talking about the PAHs being protective in some measure; is that right?

A No. Stimulating Ah receptor creates adverse

effect. It influences the ability to the cell to regulate its growth properly.

Q Does an antiestrogen cancel out an estrogenic compound?

A Yes, it would.

Q What is the relevance of the Pliskova paper to Sherrie Barnes?

A Well, I have been trying to say that, to me, it addresses the issue of the dioxin, plus the PAHs being more harmful.

Q Okay. Does it identify a particular dose or exposure level in which harm would occur?

A No. It is an in vitro study. It wouldn't have any quantitative value.

Q So it is hypothesis generating in that regard?

A No, it demonstrates in an in vitro system, a mechanism. Those give us insights into why we would have this young woman developing such a malignant cancer at such a young age following in vitro, in utero, and early childhood exposure to these chemicals.

Q Well, it looked at particular PAHs; is that right?

A Yes, they looked at two particular PAHs.

Q They did not look at creosote as a mixture?

A Right. Correct.

Q Which -- which congeners of dioxin did the Pliskova paper study?

A TCDD.

Q So you had one congener of dioxin and two different congeners of PAH?

A I don't think they actually did TCDD. They just referred TCDD studies. They, themselves, just did PAH studies.

Q Okay. So the Pliskova paper does not actually study a synergistic effect between PAHs and TCDD?

A Correct.

Q It just shows that certain -- certain PAHs at certain levels can have a dioxin-like effect?

A There is some missing page here.

Q Sorry.

A 247 is missing.

Q I will have to supply that.

A 249 is missing. 251 is missing. 252 is missing.

Q You just got the even pages?

A So I am looking for things like what they used, but the pages are missing.

Q But from the abstract -- and I apologize for that, Doctor. We will supply a full copy when we come back to this in our next session.

From the abstract, it looks like they were not studying the synergistic effect; is that right?

A No, they were not studying the synergistic effect. I am simply saying that that is one the reasons why it is relevant.

Q It suggests --

A It suggested that the two together are going to have a more likelihood of developing the cancer.

Q Can we do one more paper and then call it quits?

A We will do Revich.

MR. HOPP: Keith, you all right? Can you hang in there?

MR. PRUDHOMME: Sure.

BY MR. HOPP:

Q Let's do Revich. The next document on your list, Doctor, 20, is the Revich paper; right?

A Yea.

Q Handing you what we marked as Exhibit 150.

(Defendants' Exhibits 150 was marked for identification by the court reporter.)

BY MR. HOPP:

Q This is the Revich paper; right?

A Yes, sir.

Q And Revich is looking at dioxin exposure in

Chapaevsk, C-H-A-P-A-E-V-S-K, Russia; is that right?

A Yes.

Q What happened in Chapaevsk, Russia to make Revich want to study dioxin exposure?

A There was a pesticide plant there that made chlorinated pesticides.

Q In particular, TCDD; right?

A No. Nobody makes TCDD, but they were making lindane.

Q Lindane. Okay.

A And they generated a huge pollution with TCDD, TEQs. There was a -- they had levels that are a little bit higher than the levels that we have outside the Koppers plant in Grenada, but not too much higher. There is certainly a good overlap there.

Q Okay. All right. Did Revich find an increase incidence of breast cancer in this exposed population?

A Yes, I think that is the point. The Chapaevsk -- how did you pronounce it?

Q Chapaevsk.

A Chapaevsk women had a higher risk overall due to breast cancer. 2.1, at 1.6 to 2.7 and then some other cancers, as well.

Increase female breast cancer in all age groups compared to Russia and the Sumara region in 1998. There

is a table in here. I think a graph -- a figure that -- Figure 1 and Figure 2.

Figure 2 is the female breast cancer rate. 958 is the page. And it shows consistently at all ages higher breast cancer rate for women in that region.

Q Compared to the rest of Russia and to this other area, the Sumara area?

A Yes, which is probably the general area that this thing is in. And they also have some data on concentrations of the PCDD and PCDF in the blood, milk; soil; air; and they also have some data on how far away they were from the plant for concentration of blood.

The -- I think this is TEQ -- yes. Picogram TEQ on Table 13, they had six people that they studied which was within one to three kilometers of the plant.

Their values were 75, picogram TEQ, opposed to those that were five to eight kilometers away where it was four people. And their value was 24. And then they did some other control values.

Q So is this like a cohort study or a cross-sectional?

A This is cross-sectional, environmental, biomarker and -- yeah, cross-sectional study. I don't think they had any controls. They used, as I said already, published rates.

Q So did Revich, in looking at breast cancer, attempt to control other known risk factors?

A Let me see what he did in terms of that issue.

I think he assumed that -- I don't see that he did any analysis, for example, age of menarche, menopause, and all of that other stuff.

Q Right. Now, he did identify exposure levels or at least --

A Yes, he had exposure levels.

Q And did he identify the level at which the exposure is likely to cause harm?

A Well, I don't think we can say that because he doesn't have a no effect level.

In other words, he has a level of blood TEQs in six people that lives within one to three kilometers of the center. So we can say that if you are between the background level and that level, somewhere in there would be the level at which you start seeing any excesses.

Q He does not give us a bright line for excess levels of cancer?

A Well, what they say in regulatory circles is that he gave a single value that was the only and, therefore, the lowest observed adverse effect level of 75 in the blood.

Q 75 picograms per gram?
A Yes, picograms per gram.
Q And that is total TEQ?
A Total TEQ.
Q Which is much higher than the level that was measured in the cohort in Grenada; correct?
A Well, it is a -- it is higher than the average.
Q Total TEQ in Grenada was 34; right?
A Not the highest values, no. I think we had some others that were high.
Let's see if I can find where I did look at this. We have one -- I think one of these values. The TEQ was 92 on one of our folks and another one was 93. One had 50. One at 89. So we had some that were clearly up in that range.
Q Okay. But --
A Yeah, the mean value is whatever we said it was.
Q But what is the mean value in the Revich paper?
A 75. They didn't give the breakdown. Yeah, the mean value is higher. I agree with that. Now, there is also some soil values here.
Q Well, let's start with the workers -- female workers' blood. The workers had a total TEQ of 412; is that right?

A Yeah, that is the workers. I am talking about people who were living next to the site.

Q One to three kilometers, it was 75.2?

A That's right.

Q And for his analysis of breast cancer, does he combine the workers and women who lived near the plant or does he examine just the women who lived one to three kilometers from the plant?

A I think he may have combined them, but I don't know, looking at this. Yes, that was four workers who worked in the plant. And that he refers to an earlier paper that he published that report.

Q Six women who lived from one to three kilometers?

A And there was six women who lived between one to three kilometers. That is where the 75 came from. That's also from an earlier paper.

Q So workers from one to three kilometers combined, that is a total of ten women; right?

A Yes. Well, it does not say that they are all women; do they?

Q Yeah, look at Table 4. Female blood?

A One to three kilometers. Four -- okay. The 75 is the one -- is the six there?

Q Maybe it is a bit obscure, I mean, the title of

the table says Female Workers Blood and then the column, one to three kilometers, that is not workers. So it is a bit ambiguous; isn't it, with respect --

A Yeah, it is. That is an interesting question.

Are they all women or is this men and women? Let's see, we are talking about dioxin and public health. The guy is not a really skilled writer.

Q Well, he is Russian.

A Well, it is not his native language. It's hard for them to sometimes get it straight. Even I have had Russian papers that I read and had them translated, and they were really awful.

But here is an example of some complexity that is hard -- blood samples were taken from 14 people. 90 percent of women lived in Chapaevsk versus for more than three years. So maybe it is all women.

Q Total on Table 4 is 14. You got four workers, six --

A Yeah. 90 percent of the women -- it must be all women.

Q Okay.

A But it does not say that anywhere.

Q All right. In any event -- at any rate, Revich identifies the effect level being 75 picograms per --

A Yeah. Yeah. We do have something to look at.

MR. HOPP: Okay. All right. Shall we knock off for the day? It is 5:00 o'clock.

THE WITNESS: You won't get an argument out of me.

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I, JAMES DAHLGREN, M.D., do hereby declare under penalty of perjury that I have read the foregoing transcript; that I have made any corrections as appear noted, in ink, initialed by me, or attached hereto; that my testimony as contained herein, as corrected, is true and correct.

EXECUTED this _____ day of

_____,

20__, at _____, _____.

(City) (State)

_____

JAMES DAHLGREN, M.D.

I, the undersigned, a Certified Shorthand Reporter of the State of California, do hereby certify:

That the foregoing proceedings were taken before me at the time and place herein set forth; that any witnesses in the foregoing proceedings, prior to testifying, were placed under oath; that a verbatim record of the proceedings was made by me using machine shorthand which was thereafter transcribed under my direction; further, that the foregoing is an accurate transcription thereof.

I further certify that I am neither financially interested in the action nor a relative or employee of any attorney of any of the parties.

IN WITNESS WHEREOF, I have this date subscribed my name.

Dated: _____

_____

Diana Janniere

CSR No. 10034